MEDICAL PROCEEDINGS

MEDIESE BYDRAES

A South African Journal for the Advancement of Medical Science

'n Suid-Afrikaanse Tydskrif vir die Bevordering van die Geneeskunde

Registered at the General Post Office as a Newspaper

By die Hoofposkantoor as Nuusblad Geregistreer

Vol. 3 · No. 13 · 5s.

Johannesburg
22 Junie 1957 June 22

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IN THIS ISSUE · IN HIERDIE UITGAWE

Brandwonde in Suid-Afrika · Burns in South Africa
Burns in Children · Burns and Scalds

British Bursary for Post-Graduate Study in the United Kingdom

The attention of general practitioners registered in South Africa is drawn to page 327 of this issue, where we publish the conditions governing this post-graduate award for clinical study for a 3-month period in the United Kingdom—Editor.

Selection Committee

The members who have agreed to serve on the Selection Committee for this Bursary are: Prof. G. A. Elliott, Prof. F. Forman, Prof. S. F. Oosthuizen, Dr. H. A. Shapiro (*Honorary Chairman*), Dr. Maurice Shapiro, Dr. M. M. Suzman.

Analysis of Childhood Burns
Notes and News: Berigte
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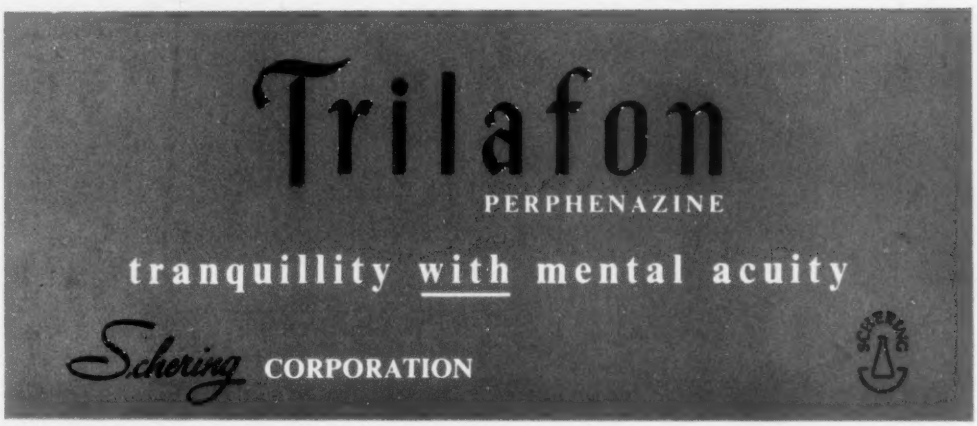

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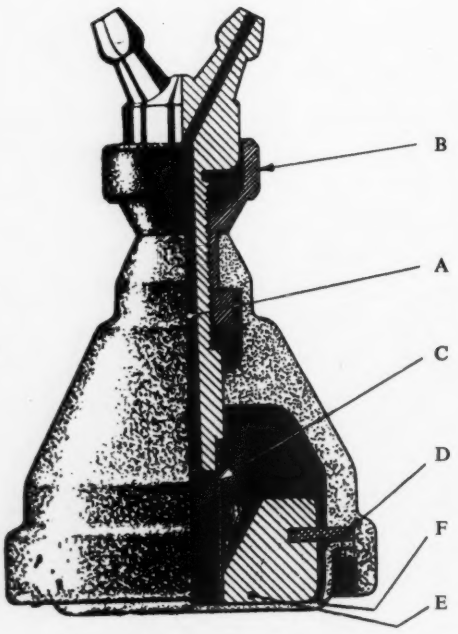

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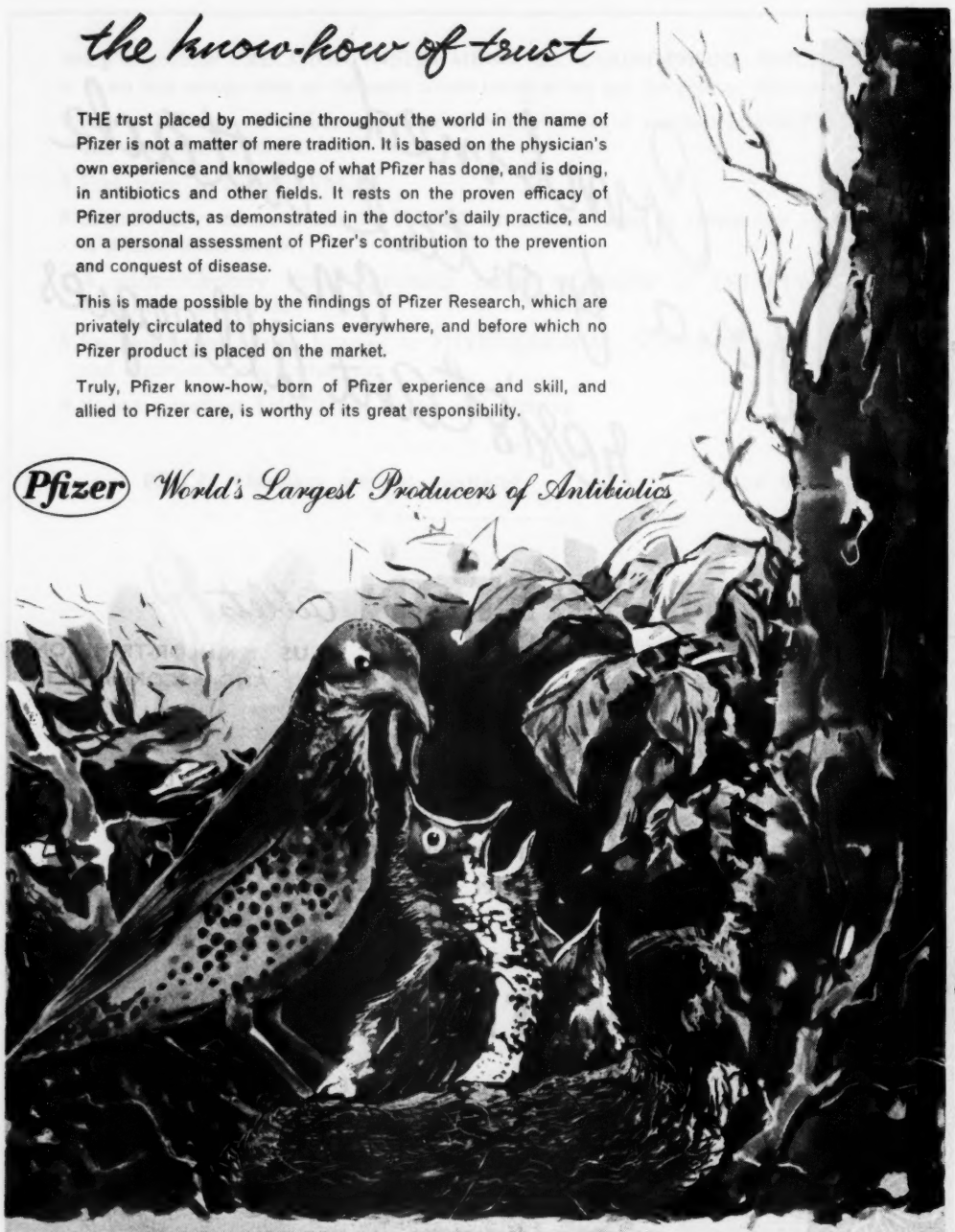

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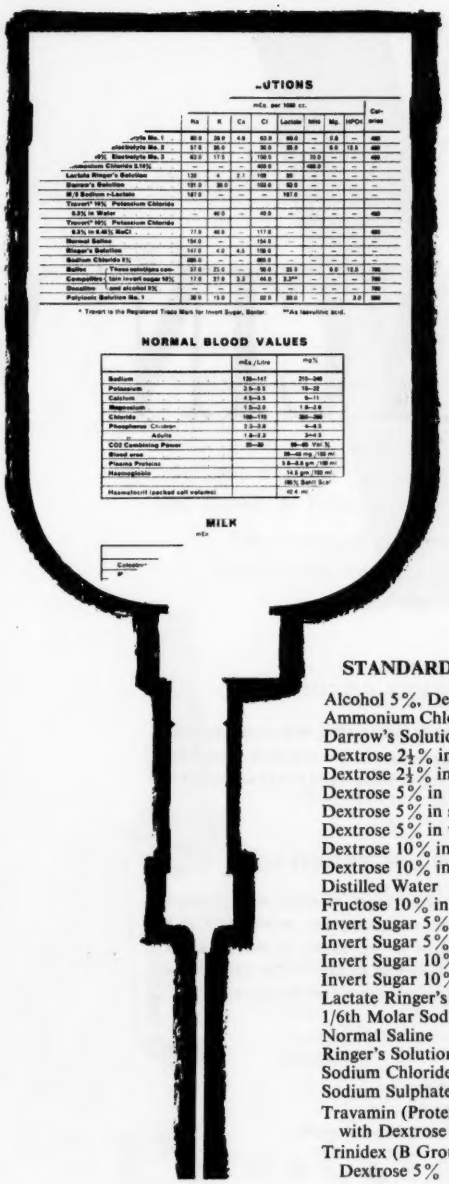

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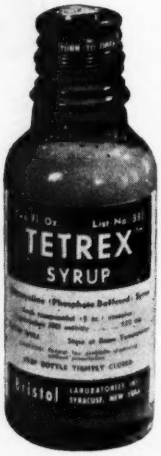

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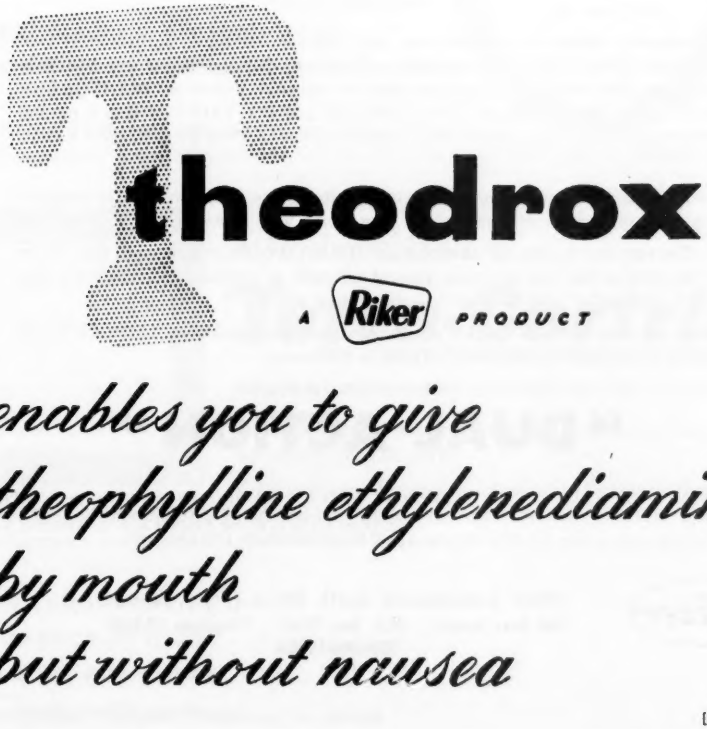

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2. LOUGHLIN E. R. and JOSEPH A. A. ANTIBIOTICS AND CHEMOTHERAPY 1:76 APL. 1951

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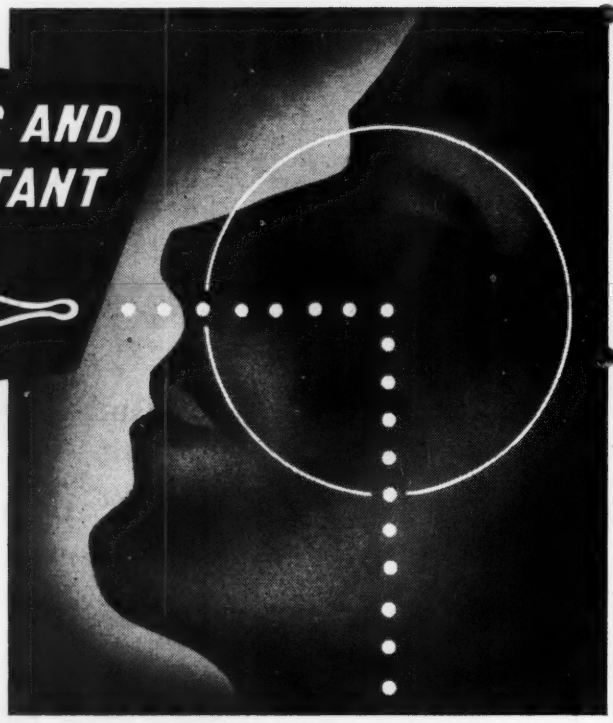

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P.O. Box 1010 · Johannesburg | Posbus 1010 · Johannesburg

Vol. 3 | 22 Junie 1957 June 22 | No. 13

REDAKSIONEEL · EDITORIAL

BRANDWONDE IN SUID-AFRIKA

VOORKOMING, BEHANDELING EN VERMINDERING VAN DIE STERFTESYFER

In hierdie land word ongeveer 30 brandwondgevalle per dag behandel, en naastenby die helfte van die slagoffers is kinders. Gemiddeld is daar ook een sterfgeval ten gevolge van brandwonde op iedere dag van die jaar.

Vergelyk dit met die sterftesyfer ten gevolge van poliomiëlitis en witseerkeel. In 1952 was daar 331 sterfgevalle ten gevolge van difterie. Selfs die poliomiëlitis-epidemies van die afgelope paar jaar, met hul sterftesyfer van 2-5% (na gelang van die erns van die uitbreking) het minder menselewens opgeëis as brandwonde. Gedurende die afgelope paar jaar is meer as 100 persone jaarliks ten gevolge van poliomiëlitis oorlede in vergelyking met 'n brandwond-sterftesyfer van meer as 370[1] onder alle rasse in Suid-Afrika. Van laasgenoemde getal was 308 nie-blankes.

Hierdie toestand is nie eie aan Suid-Afrika nie. Die aantal mense wat in die Verenigde State ten gevolge van brandwonde gesterf het, was twee maal groter as die aantal poliomiëlitis-slagoffers in 'n nie-epidemiese jaar.[2]

Net soos in die geval van poliomiëlitis en witseerkeel, is die slagoffers van brandwonde meestal kinders. In Engeland en Wallis was 40% van die brandwondgevalle wat gedurende die jare 1935-39 in die hospitale behandel is,

1. Buro van Sensus en Statistiek, Pretoria (1956): Private mededeling.
2. Owens, N. (1944): Surg., **15**, 207.

BURNS IN SOUTH AFRICA

PREVENTION, TREATMENT AND REDUCTION OF MORTALITY

In this country there are probably some 30 cases of burns treated daily, about half of them being in children. There is also, on the average, one death from burns for every day of the year.

Contrast this with the mortality from poliomyelitis and diphtheria. In 1952 there were 331 deaths from diphtheria. Even the poliomyelitis epidemics of the last few years, carrying a death rate of 2—5% (according to the severity of the outbreak), caused fewer total deaths than did burns. More than 100 deaths annually from poliomyelitis during the last few years contrast with an annual mortality from burns for all races in South Africa of over 370,[1] of which 308 were non-White.

The situation is not peculiar to South Africa. Twice as many people died from burns in the United States as were afflicted with poliomyelitis in a non-epidemic year.[2]

Like poliomyelitis and diphtheria, burns affect mainly children. In England and Wales, 40% of burns admissions (1935-1939) were children,[3] and nearly half the annual death rate from burns involved children under 10 years. In a series reported elsewhere in this

1. Bureau of Census and Statistics, Pretoria (1956): Private communication.
2. Owens, N. (1944): Surg. **15**, 207.
3. Colebrook, L. (1946): Monthly Bull. Minist. Hlth. (Lond.). Page 214.

kinders[3]; en van die totale aantal mense wat jaarliks ten gevolge van brandwonde sterf, is byna die helfte kinders onder 10 jaar. In 'n reeks waaroor elders in hierdie uitgawe verslag gedoen word, was 40-55% kinders.

Die tragedie van hierdie syfers lê opgesluit in die feit dat baie van hierdie ongelukke, veral vir sover kinders daarby betrokke was, voorkom kon gewees het as volwassenes net groter sorg en bedagsaamheid aan die dag gelê het. Uitgebreide en duur navorsingsinrigtings is nie nodig om die aantal brandwondslagoffers te verminder nie.

Byna alle brandwonde is die gevolg van ongelukke, en in die geval van kinders is dit altyd die geval. Onbeskermde vure, elektriese verwarmers en ontvlambare nagjurkies en tabberds hou 'n dodelike gevaar vir jong kinders in.[4] Hul natuurlike nuuskierigheid loop dikwels uit op brandwonde wat veroorsaak word deur die inhoud van kastrolle en koppies wat omgekantel word. Waar dit enigsins moontlik is, moet kinders nie in die kombuis toegelaat word nie. Simson[7] het onlangs aangetoon dat twee-derdes van die brandwonde by jong blanke kinders deur warm vloeistowwe veroorsaak word, en 'n dergelike syfer is van toepassing op nie-blanke kinders in Durban. Warm water, of dit nou al in 'n bad of 'n emmer is, bly altyd gevaarlik. Ouers is wel deeglik bewus hiervan, maar besef dikwels nie watter verskriklike ongelukke warm water kan veroorsaak nie—totdat so 'n ongeluk plaasvind. Onder nie-blankes wat in armoedige en oorvolle huise woon, hou die lamp wat maklik omslaan, die blikke kookwater, en die groot oop kastrolle die grootste gevaar in.

Die sterftesyfer toon slegs gedeeltelik aan wat hierdie soort ongelukke die gemeenskap kos. Die lyding, die ontsiering en die ongeskiktheid voortspruitende uit sepse en kontrakture is onder die ergstes wat in chirurgiese sale aangetref word.

Wat dit betref, is die opvoeding van die publiek 'n dringende vereiste. Ons behoort 'n kragdadige publisiteitsveldtog van stapel te stuur—net soos die padveiligheidsorganisasies wat gebruik maak van rolprente, advertensies en biljette in openbare kantore en skole. Verstandige wetgewers kan miskien oorreed word om te help deur aan te dring op doelmatige skerms vir alle elektriese verwarmers en toestelle. Duidelike instruksies en waarskuwings is verrassend doeltreffend, selfs in die geval van nie-blanke moeders, veral as dit hulle op die hart gedruk word dat hulle veel pyn en lyding kan voorkom as hulle net 'n bietjie bedagsaamheid en sorg aan die

3. Colebrook, L. (1946): Monthly Bull. Minist. Hlth. (Londen). Bladsy 214.
4. Becker, J. M. en Arts, C. P. (1956): A.M.A. Arch. Surg., **73**, 207.
7. Simson, J. (1956): S. Afr. Tydskr. Geneesk., **30**, 1030.

issue, 40-55% were children.

The tragedy of these figures is that these accidents, particularly in children, could largely be prevented if greater care and consideration were exercised by adults. Extensive and expensive research institutions are not needed to reduce the number of cases of burns.

Nearly all burns are accidental and certainly all are so in the case of children. Unguarded fires, electric heaters and inflammable nightgowns and dresses contribute a lethal threat to young children.[4] Their natural curiosity results in scalds from overturned pots and cups. Where possible, kitchens should be out-of-bounds to children. Recently Simson[7] showed that over two thirds of burns in young White children were due to hot liquids, and a similar figure is true for non-White children in Durban. Any hot water, whether in a bath or a bucket, is potentially dangerous. Parents are well aware of these facts but seldom foresee the dreadful accidents that may happen until it is too late. Amongst non-Whites living in poverty-stricken and overcrowded homes, the easily overturned lamp, the tins of boiling water and the large open cooking pots are the greatest menace.

Mortality rates illustrate only a part of the cost to the community. The suffering, disfigurement and disabilities from sepsis and contractures are amongst the worst seen in surgical wards.

Public education is a real necessity in this respect. We need to adopt a vigorous publicity campaign similar to that of the road safety organizations, which use films, advertisements and posters in public offices and schools. Wise legislators may be persuaded to help by insisting on efficient standards of guards for all electric fires and appliances. Careful instructions and warnings are surprisingly effective, even with non-White mothers, when they are made to understand the pain and suffering they themselves can avert with a little thought and care. Such steps are essential in impressing on the members of the public the fact that they themselves are personally and directly responsible for the appalling loss of life of their own children in their own homes.

The treatment of burns represents a major economic factor in surgical admissions to a hospital. Common surgical conditions such as those requiring abdominal and chest opera-

4. Becker, J. M. and Artz, C. P. (1956): A.M.A. Arch. Surg., **73**, 207.
7. Simson, J. (1956): S. Afr. Med. J., **30**, 1030.

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dag lê. Sodanige stappe is noodsaaklik as ons dit by lede van die publiek wil tuisbring dat hulle self persoonlik en regstreeks verantwoordelik is vir die ontstellende lewensverlies onder hul eie kinders in hul eie huise.

Die behandeling van brandwonde is 'n eerste-rangse ekonomiese faktor wat betref die aantal chirurgiese gevalle wat in ons hospitale opgeneem word. Mense wat chirurgie nodig het, byvoorbeeld dié wat buik- of borsoperasies moet ondergaan, en die meeste traumatiese gevalle bring tussen 14 en 28 dae in die hospitaal deur—ongeveer net so lank soos die hospitaalverblyf van brandwondslagoffers met oppervaktes van minder as 15% met gedeeltelik dikte-verlies. In ernstiger brandwondgevalle waar 'n gedeelte en soms die meeste van die vel volkome vernietig is, styg die hospitaalverblyf tot 50-70 dae in die geval van diegene met 20-30% brandwonde, en 60-180 dae in die geval van diegene met groter brandoppervlaktes.

Die koste per pasiënt per dag in hospitale wissel van 25s. in sommige hospitale vir nie-blankes tot meer as £4 per dag in die hospitale vir stedelike blankes. Met die steeds toenemende vermindering van die brandwond-sterftesyfer is die duur van die pasiënt se hospitaalverblyf geneig om te styg, liewer as om te daal. Gesien die vooruitgang wat daar op die gebied van mediese kennis gemaak word, is dit 'n buitengewone verskynsel. Afgesien van die langer verblyf in die hospitaal, is dit ook 'n feit dat die veelvuldigheid van prosedures en die duur verbande wat herhaaldelik op ingeplante oppervlaktes aangelê moet word, die koste tot twee maal die gemiddelde syfer verhoog. By die King Edward VIII-hospitaal in Durban is daar bv. bereken dat die koste van die hospitaalbehandeling van kinders wat die lewe behou, op ongeveer £10,000 per jaar te staan kom. En hoe meer lewens gered word, hoe hoër styg die koste. So is daar verder bereken dat 'n addisionele £10,000 aan hierdie ekstra aantal oorlewendes bestee sal moet word.

Met die jongste vorderings op die gebied van die verbetering van die vloeistofewewig en die herstel van die bloedvolume met behulp van plasma en bloedoortappings het die sterftesyfer ten gevolge van brandwonde aansienlik afgeneem.[5] Die tans beskikbare middele om infeksie te bestry is ongetwyfeld die grootste bydraende faktor tot die vermindering van die onmiddellike en latere sterftesyfer; maar die antibiotica het min effek tensy die allersorgvuldigste aandag aan asepse bestee word. Die veronagsaming van chirurgiese beginsels wat plaasvind iedere keer as 'n persoon lydende aan 'n oop wond—en dit is wat brandwonde in werklikheid is—tot 'n algemene chirurgiese saal toegelaat word, stem ooreen met 'n operasie aan 'n lit in 'n ongevalle-afdeling. Geen behandeling, hoe goed dit ook al is, kan vergoed vir die skade wat gedurende die eerste paar uur aangerig word nie. Colebrook se beskrywing van die toelating van 'n brandwondpasiënt in 'n algemene hospitaal word nog veels te dikwels teëgekom:[6]

‚Die kind word eers in die Ongevalle-afdeling ondersoek, miskien aanvanklik deur 'n junior chirurgiese amptenaar wat dan in verbinding tree

5. Bull, J. P. en Fisher, A. J. (1954): Ann. Surg., **139**, 3.
6. Colebrook, L. (1950): *A New Approach to the Treatment of Burns and Scalds.* Fine Technical Publications.

tions, and most traumatic cases, spend 14-28 days in hospital—about the same time as spent by those cases of burns with areas under 15% with partial thickness loss. In major burn cases, which usually have some, if not most, skin fully destroyed, the length of stay jumps up to 50-70 days for 20-30% burns, and 60-180 days for larger areas.

The cost per patient per day in hospital ranges from 25s. in some non-White hospitals to over £4 a day in urban White hospitals. With the increasing reduction in the mortality figures of burns, the hospital stay lengthens rather than shortens. This is not usually the case with advances in medical knowledge. Apart from a longer hospital stay, the multiplicity of procedures and the expensive frequent dressings to large grafted areas raise this cost probably to double the average figure. It has been estimated that at King Edward VIII Hospital, Durban, the hospital cost of children who survive is about £10,000 a year. With an increased survival rate, the costs ascend steeply, and it has further been estimated that an additional £10,000 would have to be spent on this extra number of survivors.

With recent advances in the correction of fluid balance and the restoration of blood volume by plasma and blood transfusion, the immediate mortality of burns has been considerably reduced.[5] The means of combating infection which are now available represent the biggest contributory factor in the reduction of immediate and late mortality figures; but antibiotics are of little effect unless scrupulous attention is paid to asepsis. The breach of surgical principles in admitting open wounds —which burns are—to the general surgical ward, is akin to operating on a joint in a casualty department. No treatment, however modern and thorough, can ever undo the damage done in the first few hours. Colebrook's description of the admission of a burnt patient to a general hospital still occurs all too frequently:[6]

'The child is seen in the Casualty Department, perhaps first by a junior surgical officer who communicates with the house surgeon who is admitting that day (this may occupy 15 minutes) and then sends the child to the ward. The surgeon into whose bed the child is admitted is perhaps not in the hospital or is busy on a list of operations, so he does not see the child for several hours, or possibly not until the following day . . .'

5. Bull, J. P. and Fisher, A. J. (1954): Ann. Surg., **139**, 3.
6. Colebrook, L. (1950): *A New Approach to the Treatment of Burns and Scalds.* Fine Technical Publications.

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met die inwonende chirurg wat op daardie besondere dag pasiënte toelaat. Dit kan 15 minute in beslag neem. Dan word die kind na 'n saal gestuur. Die chirurg, in een van wie se beddens die kind geplaas word, is miskien nie in die hospitaal nie, of is besig met 'n reeks operasies. Etlike ure verstryk dus voordat hy die kind ondersoek. Moontlik doen hy dit eers die volgende dag. . . .'

(Die inwonende praktisyn) ,wat slegs 'n paar maande tevore gekwalifiseer en miskien 'n paar weke gelede aangestel is, het geen vorige ondervinding van 'n ernstige brandwond nie. Hy besef derhalwe nie dat die kind se lewe afhang van die vinnige vervanging van vloeistof om skok te bestry nie. Hy verbind die kind se brandwonde inderhaas, of, as daar ander belangrike pligte op hom wag, gelas hy die saalsuster om dit te doen. Die verbinding geskied in die oop saal—miskien slegs 'n paar voet vanaf 'n ander pasiënt met 'n beseerde gesig wat besmet en slegs gedeeltelik bedek is.

Dit is 'n besige saal, en geen verpleegster kan afgesonder word om by die kind te bly om die afdrup te reguleer, om by herhaaldelike tussenpose klein teugies water mondeling toe te dien, en om die pols, die temperatuur en die asemhaling al om die halfuur aan te teken nie.'

Dit is duidelik dat doeltreffende asepse bewerkstellig kan word alleen deur algehele isolasie deur middel van 'n georganiseerde stelsel wat onder meer voorsiening maak vir 'n vertrek vir die toelating van brandwondslagoffers in 'n Ongevalle-afdeling, en die onmiddellike verplasing van die pasiënt na 'n saal wat afgesonder is van die sepse van algemene sale. So 'n brandwond-eenheid behoort 'n noodsaaklike kenmerk te wees van enige hospitaal waar meer as 100 brandwondgevalle per jaar behandel word. Nêrens het dit duideliker aan die lig gekom nie as in die Birminghamse Ongelukshospitaal waar die hoeksteen van suksesvolle behandeling is om die pasiënt dadelik van alle ander pasiënte af te sonder en om sy wonde met die volledige rituaal van asepse te behandel.

Hoe lang die pasiënt in die hospitaal sal moet bly, hang regstreeks af van die mate van infeksie in die verbrande oppervlakte. Beheer van infeksies beteken vroeë inplanting en vroeë rehabilitasie, en—van ewe groot belang—dit verlig die pyn en verban die vrees vir die verbinding van die wonde.

Die gegewens wat by die King Edward VIII-hospitaal ingewin is, staaf die bewering dat 'n beperking van sepse nie alleen die hospitaalkoste verminder nie, maar ook lewens red.

Daar is ook aangetoon dat dit besonder voordelig sal wees as alle ernstige brandwondgevalle uit die nabygeleë streke in so 'n sentrale eenheid opgeneem kan word. Die gevaar verbonde aan 'n reis wat 'n halfuur langer duur, is nie so groot soos die gevare verbonde aan hastige toelating tot 'n klein hospitaal wat sleg toegerus is om ernstige brandwonde te behandel.

Verpleegsters wat opgelei is in die behandeling van brandwonde is 'n belangrike vereiste vir die instandhouding van doeltreffende asepse en die algemene welsyn van die pasiënt. Die verbinding van brandwonde in ongevalle-afdelings deur 'n opgeleide personeel verminder die aantal pasiënte wat tot die hospitaal toegelaat moet word. Insgelyks kan opgeleide verpleegsters in die navolgingsafdeling minder ernstige brandwonde behandel wat andersins miskien hospitaalbehandeling nodig sal hê.

In 'n aanvanklike proefneming in Suid-Afrikaanse stede kan 'n sentrale eenheid, bestaande uit beddens

(The house surgeon) ' having been qualified only a few months and appointed only a few weeks before, has had no previous experience of a severe burn. He does not therefore realize that the child's life depends on speedy replacement of fluid to combat shock. He hastily dresses the child's burns or, if his other duties are pressing, instructs the ward sister to do so. The dressing is done in the open ward, perhaps only a few feet away from another patient whose face has been injured and is now infected and only partially covered.

The ward is very busy and no nurse can be spared to remain with the child to regulate the drip, to give it sips by mouth at frequent intervals and to keep a half-hourly chart of pulse, temperature and respiration.'

It is apparent that efficient asepsis can only be attained by complete isolation through an organized system which must include a burns admission room in a Casualty Department and immediate transfer to a ward separated from the sepsis of general wards. Such a Burns Unit is an essential feature in any hospital admitting more than 100 burns cases annually. Nowhere has it been shown more forcibly than at the Birmingham Accident Hospital that the crux of successful treatment is to isolate the patient immediately from all other patients and to handle his wounds with the complete ritual of asepsis.

The length of stay in hospital is directly connected with the degree of infection of the burnt area. Control of infection means early grafting and early rehabilitation and, no less important, eases pain and consequently removes the terror of dressings.

The data quoted for the King Edward VIII Hospital support the contention that a limitation of sepsis would reduce hospital costs as well as save lives.

It has been shown further that it is of considerable advantage for such a central unit to admit all major burn cases from surrounding regions. It is better to risk the extra half hour of travel than to permit hasty admission to a small hospital, ill-equipped to deal with major burns.

Nurses trained in the care of burns are important for maintaining proper asepsis and the general welfare of the patient. Casualty dressings of burns, by trained personnel, limits hospital admissions. Similarly, in the follow-up department, trained nurses can handle minor degrees of burns which would otherwise require hospital treatment.

In South African cities, in a pilot experiment, one such hospital could house a central unit comprising beds for about 20 adults and 10 children. The cost of establishing such centres is not prohibitive. Single storey build-

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vir ongeveer 20 volwassenes en 10 kinders, by so 'n hospitaal ingerig word. Die koste verbonde aan die instelling van sodanige sentrums is nie onmoontlik hoog nie. 'n Enkelverdiepinggebou met kamerakkommodasie en 'n verbandvertrek, en met lugreëling in ten minste sommige van die kamers, kan teen 'n matige aanvanklike koste opgerig word. En vir hierdie uitgawe sal die hospitaal ruimskoots vergoed word deur die vermindering van die koste wat hierdie soort ongelukke meebring.

'n Brandwondeenheid behoort 'n essensiële kenmerk te wees van enige moderne hospitaal waar letsels behandel word. In sulke omstandighede kan die behandeling sorgvuldig beplan word, iets wat glad nie so maklik is nie wanneer hematokrietberekenings elke uur gedoen en die binne-aarse vloeistof-opneming gekontroleer moet word, en wanneer dit die verpleegpersoneel op die hart gedruk moet word dat 'n operasiesaal-rituaal steeds 'n lewensbelangrike vereiste is. Buite-hospitale sal aangemoedig word as hulle weet dat so 'n organisasie tot hul diens is. Dit is skaars nodig om by te voeg dat, net soos in alle spesiale vertakkinge van die chirurgie, die oplossing van tot dusver onopgeloste probleme makliker gevind kan word deur ervare sentrale organisasies.

ings with cubicle accommodation and a dressing room, and with at least some of the rooms air-conditioned, would entail a moderate initial cost. This would be amply repaid in lessening the burdensome cost of these injuries to hospitals.

A Burns Unit represents an essential feature of a modern hospital dealing with trauma. Under these conditions, treatment can be carefully guided, not an easy task when hourly haematocrit estimations and intravenous fluid intake must be controlled and the nursing staff brought to realize the constant need of a theatre ritual. Peripheral hospitals are encouraged in the knowledge that such an organization is at their service. It is hardly necessary to add that, as in all special branches of surgery, solutions to as yet unresolved problems may sometimes be reached more rapidly by skilled central organizations.

BURNS IN CHILDREN

THE PRINCIPLES OF TREATMENT

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Burns comprise an important group of surgical admissions in children in large urban communities. Most of the accidents occur as the result of parental carelessness or thoughtlessness in the home. Attention has previously been drawn[24] to the considerable medical and administrative advantages of having the organization of the care and treatment of these injuries centralized in a Burns Unit of a general hospital.

The problem in the treatment of burns is that of the care of open wounds which, beyond a certain definite area in extent, are associated with a distinct type of surgical shock and which frequently require early surgical repair. These three features will be discussed in detail.

FIRST AID MEASURES

The aim of first aid treatment is to prevent shock and, by keeping the burnt area covered and clean, to avoid infection.

Despite the variety of ointments and special dressings available, the simplest and safest advice to the parent faced with this emergency is to cover the burn with a clean pillow case or sheet and seek medical aid. The ideal is to keep the burnt part dry and covered.

The pain of burns is not always severe and the child's upset condition is often due largely to fright. Deep burn areas are insensitive but partial thickness loss of skin does cause intense stinging. If the child finds the pain of a limb burn intolerable, the sting can be relieved by running cold tap water and the part can then be wrapped in a double sheet to keep the outer layer dry. All burnt clothing or shoes should be left on until the child reaches hospital. A sedative should be prescribed and the child kept warmly wrapped and given drinks.

ASSESSMENT OF THE BURNT SURFACE

(*a*) *Extent of the Surface Area Burnt.* It is essential to make only one accurate examination of the burnt areas so as to reduce to a minimum the chance of infection by avoiding touching or breathing on the exposed part. A

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burn deemed suitable for home treatment should be handled with the same caution and efforts made to preserve an aseptic method. Any burn larger than 2% (approximately twice a patient's hand size) is better dealt with in the Burn Admission Room of a hospital.

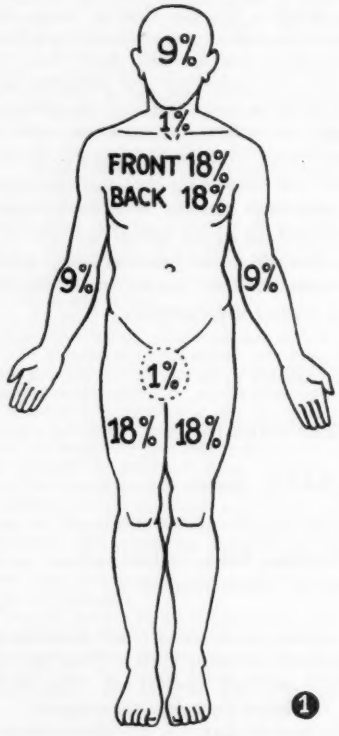

Fig. 1. Wallace's 'Rule of Nine'. (With acknowledgments to the author).

Allowing for variations in size and age, Wallace's 'Rule of Nines' is a sufficiently accurate and simple guide[3, 22] (Fig. 1) and is based on the following figures:

Head and neck = 9%;
1 upper limb = 9%;
1 lower limb = 18%;
Anterior chest and abdomen = 18%;
Back and buttocks = 18%;
Perineum = 1%.

This system of assessment is designed essentially for, and is more accurate in, adults. In children there are proportionate differences. Lund and Browder[16] have devised an accurate table which is useful for reference (Table 1). In young children the head is relatively greater and the lower limbs relatively less in proportion than in adults. With experience, the child's variations are readily remembered and these charts should be available in the burns reception room for ready reference.

A rapid assessment can be made when large areas are burnt; in the case of smaller or scattered areas, there ar etwo further easy aids;

i. The size of the patient's hand (palm and fingers) represents 1% surface area; this is a particularly valuable method for measuring irregularly shaped burn areas of limbs:

ii. In the case of large burns of the abdomen, back and chest it is often simpler to estimate the area left unburned and so measure the extent.

TABLE 1

Age	*Birth*	1	5	10	15	*Adult*
Area						
Head	19	17	13	11	9	7
Neck	2	2	2	2	2	2
Anterior trunk*	13	13	13	13	13	13
Posterior trunk†	13	13	13	13	13	13
Buttocks	5	5	5	5	5	5
Genitalia	1	1	1	1	1	1
Upper arms	8	8	8	8	8	8
Forearms	6	6	6	6	6	6
Hands	5	5	5	5	5	5
Thighs	11	13	16	17	18	19
Legs	10	10	11	12	13	14
Feet	7	7	7	7	7	7
Total	100	100	100	100	100	100

*Without neck or genitalia.
†Without neck or buttocks.

(*With acknowledgements to Lund and Browder*[16])

There are important exceptions to the use of these figures, particularly in burns of contiguous areas of the neck and supraclavicular region, large pendulous breasts and fatty buttocks, which should be estimated by hand-size, as they are often considerably greater in extent than is indicated on the chart.

Erythema must not be taken into consideration in measuring the extent of the burn as it is of little consequence in treatment and considerably alters the estimation.

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(*b*) *Site.* Certain areas of the body pose special problems, either because they produce a greater degree of shock, require skilled nursing or because the disability that may result is crippling.

Any burn of the face, mouth or hand should be admitted to hospital, no matter how small the extent, as vital function may be irreparably damaged; eyesight is endangered and lips, eyelids and nose require careful local toilet to minimize infection and reduce the need later for complicated plastic repairs. Gross swelling of the face with closed eyes and nares blocked by exudate is common. Facial burns, like those of the abdomen and the perineum, produce a state of shock out of proportion to the extent burnt. Burns of the mouth and air passages may require urgent tracheotomy to save life.

Unless fingers are dressed separately and treated in a position of function, followed by grafting at the earliest possible time, disabling contractures may follow and require amputation. Interphalangeal joints thinly covered by skin are easily exposed with what would be only deep partial burns in other parts and, if so, this usually leads to ankylosis.

The perineum is particularly sensitive and pain is a prominent feature; chafing surfaces require constant attention to prevent maceration and avoid faecal contamination and nursing is simplified by a gallows suspension.

(*c*) *Depth.* The common practice of assessing burns as 1st, 2nd and 3rd degree, etc., the Dupuytren classification, is often fallacious on early inspection. As a descriptive classification distinguishing between reddening, blistering and charring it is of limited value in initial treatment, as blistering may occur in a previously erythematous area 3 or even 4 days later, while full thickness loss is often preceded by a blistered stage. Its main use, distinguishing those areas which are deeply burnt and thus indicating the degree of local red blood cell destruction, is equally well considered in other terms. Often an assessment such as '1st degree burns, 10% area' is finally found to be mainly full thickness loss and shock may be left untreated.

The only clinical distinction that can be made is partial thickness skin loss and full thickness skin loss. Blistering usually indicates the former, while charring and a dead white appearance are typical of the latter. Full thickness loss is not always easy to differentiate in the early stages, and of the many clinical and dye tests used, such as van Gieson's stain or intravenous fluorescin, the most useful is the response to pin prick in the burnt area.[15] Partial thickness loss is sensitive to evenly applied pin prick pressure, while full thickness skin loss is anaesthetic. This is not always true of specialized areas like the palm, face and scalp, where very sensitive areas may lose sensation yet heal satisfactorily without grafting. On the other hand, thick calloused skin, as on the sole, may lose sensation and yet be only thinly destroyed. Recent experimental work indicates that neural degeneration is only partly complete after 4 hours, nearly complete in 24 hours, and progresses during the first 4 days. Re-innervation proceeds slowly, and is still not complete by 7 weeks.[12]

This clinical distinction emphasizes the main considerations in the treatment of burns, the need for colloid and fluid replacement in partial thickness burns and the earliest possible replacement of skin in full thickness loss. The extent of local red cell destruction in the latter makes repeated haemoglobin estimations and blood replacement an essential feature of early care and pre-operative treatment.

In general, scalds tend to cause partial thickness skin loss, as the hot fluid, which is not usually at boiling point, runs off immediately; but this is by no means true for the delicate skin of a child. Hot oils which retain their heat tend to destroy the full thickness of the skin. Frequently it is only at the end of 10 days that the final distinction can be made, when partial thickness skin loss will show progressive healing while full thickness loss will be starting to separate. Fig. 2 shows diagrammatically the level at which partial thickness skin loss occurs, indicating that skin can still regenerate from deeply placed sweat glands and hair follicles.

The greatest difficulty is the differentiation between deep partial loss (where only pearly islands of regenerating epithelium remain) and superficial partial loss (which, by 10-12 days, will show almost complete shiny delicate epithelium replacement). Many deep partial thickness burns require grafting, partly to prevent granulation tissue from overwhelming small scattered areas of new skin and also to act as a stimulant and dressing for healing areas.

The clinical application of the assessment of the extent of the burn depends on the established observation that a 10% burn in a child will usually produce surgical shock, and require intravenous fluid infusion for a least 24 hours to replace colloid and fluid loss. A 10% burn is equivalent approximately to one upper limb in extent. It is this important guide

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which underlines the need for prompt measures against shock.

It is safest to send any burnt child to hospital when the extent of the injury is greater than 2% surface area; it may not be necessary to admit such children and adequate treatment may be given in the burn receiving room and the child seen regularly as an out-patient and admitted when necessary for repair of any full thickness loss that becomes evident some days later.

In summary, the indications for admission of a burnt child to hospital are:

1. All burns of 10% surface area or more.
2. All burns of face, hands and perineum.
3. Chemical, flash and electrical burns.
4. Deep burns of any size with skin destruction.
5. Partial thickness burns greater in extent than 2% which, after treatment, can often be sent home.

TREATMENT OF BURNS IN CHILDREN

(*a*) SMALL BURNS SUITABLE FOR OUT-PATIENT TREATMENT

After assessing that a 2-3% partial thickness burn of a limb is suitable for home or out-patient treatment, the child is given a sedative such as Inj. Heroin (gr. 1/96-1/12 according to age)[21] or Nepenthe (1 m. per year of age). Phenobarbitone often excites a child already anxious and restless, and has a dangerous cumulative effect. The burnt area is gently cleaned with a detergent such as Cetrimide 1% and any blisters snipped and loose cuticular sheets peeled off. In the case of a limb, dry gauze dressing or penicillin cream on gauze and a liberal amount of cotton wool are applied, firmly held as a pressure occlusive dressing by crepe bandage extending well above and below the area and strapped to the skin at the two margins. A light plaster shell can be added and the limb elevated by a sling or pillows in bed (Fig. 3).

Small children need handbags to prevent their picking at the dressings (Fig. 4). In addition arms can be held extended in plaster of Paris casts.

The dressing is undisturbed for 8-10 days unless the child has a fever, the dressing soaks through, smells (indicating infection) or if the dressing works loose. At the end of 10 days it may be soaked off in a bath and any new

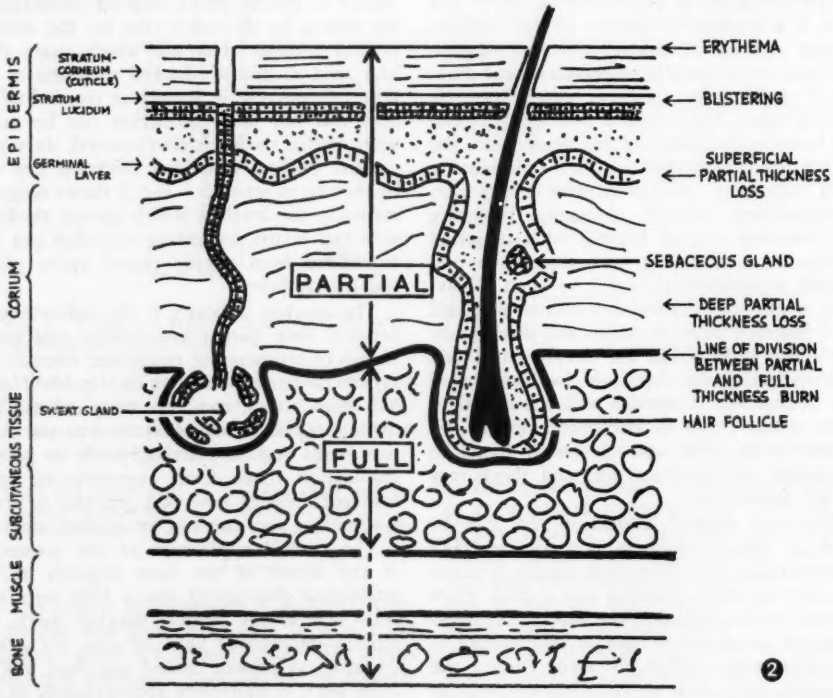

Fig. 2.

Fig. 3. Plaster shells over occlusive dressings for burns of the upper limb.
Fig. 4. Hand bags to prevent picking at burnt areas.

skin will then be visible. A dry dressing for a few more days is necessary for protection. If there is any doubt about progress or fear of commencing contractures of joints the child should, if being treated as an out-patient, be admitted to hospital.

Finally, it is worth checking the haemoglobin level even as an out-patient in 5 or 6 days, as a reduced level is often the result of infection and the cause of poor healing.

(*b*) BURNS ADMITTED TO HOSPITAL

i. *Under* 10% *Surface Area Burn.* Burns of limbs under 10% area unassociated with shock are dealt with in the same fashion by occlusive pressure dressings. In addition to hand bags which are necessary in small children, precautions must be taken to prevent soiling of adjacent burns by urine or faeces. When the upper thighs are burnt the legs should be abducted on a splint cushioned by wool, or by a gallows suspension. Burns of the perineum are sprinkled with penicillin powder in a lactose medium to give a dry coating, the treatment modified by Wallace for other parts of the body as the exposure method.[22, 23] The powder is applied 2-hourly until a crust is formed. Crusts tend to crack and require constant attention to make sure that moist areas or cracks have powder re-applied. Pus may pocket under crusts, which should then be soaked off with saline to allow drainage. Otherwise the crust is soaked off after 10 days and the depth of the skin burn then determined.

The eyes and conjunctivae are protected by Albucid drops and castor oil drops, and facial burns covered with penicillin powder. If the scalp is burnt hair is cut short on and around the area; but if not, the hair should be kept in place by a surgeon's cap or scarf.

Digits require separate firm pressure dressings in the position of function and finger movements are encouraged from the start.

Small linear or oval full skin loss areas due to chemical or electrical burns should be treated by primary excision with suture or covering by a split skin graft.[13]

ii. *Over* 10% *Surface Area Burn.* Burns over 10% surface area will require intravenous fluids for at least 24 hours, no matter how well the patient may seem on admission. The urgency with which major burns are resuscitated will determine how rapidly the child recovers.

Burn shock, while similar in some respects to the oligaemic shock of haemorrhage, trauma or a sudden abdominal catastrophe, is in its onset and subsequent development very different with special features of its own. Frequently the patient not only appears well with a good steady pulse and warm limbs, but the

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bright mental state belies the severity of the condition until 8-10 hours later the picture of advanced and often irreversible shock with low blood pressure, rapid pulse and restless exhaustion supervenes.

The loss of colloid and fluid is gradual and adaptation occurs in the early hours; only when this redistribution of the body fluids is not sufficiently aided by replacement does the typical picture of oligaemic shock occur.[6]

As soon as the patient is admitted to the ward, a bed is prepared with a sterile bed sheet, upon which sterile gamgee is laid and penicillin powder sprinkled. While the patient is still on the stretcher, and with attendants masked and gowned, all clothes are removed and the child gently lifted on to the sterile bed. During this procedure the opportunity is taken of assessing rapidly and accurately the area burnt and this is immediately recorded on a pictorial burn chart. This should be the only disturbance of the patient, as any further rolling and turning will aggravate the shocked state. An analgesic is given at once using either heroin or nepenthe, and antibiotic treatment is started.

The patient is covered by a sterile sheet, suspended by 3 or 4 cords stretched between two high bed ends so that the sheet does not come into contact with the body; alternately the sheet is suspended under cradles.

No toilet or dressing is allowed until such time as the patient has recovered from the state of shock.

Intravenous fluid replacement is urgently required to control shock and no more than half an hour should elapse after admission before the infusion is started. The same urgency is required as for sharp haemorrhage and it needs constant vigilance to see that the infusion, once started, continues. Often the first few hundred ml. tend to run in slowly and valuable time is lost. It should be arranged with a cannula or polythene tubing tied into a vein. A needle puncture technique is satisfactory in older children, using a trocar needle and cannula of the Guest pattern and the limb immobilized by extension in a plaster cast. In extensive burns, especially those involving ankles and elbows, a polythene catheter is inserted into the saphenous vein in the groin, if necessary cutting through burnt skin. This ensures a reliable and continuous infusion for some days, if necessary.

Immediately on admission pulse and temperature charts and fluid balance records are started by the nurse in charge. Blood pressure readings are frequently impossible to obtain but should be recorded if an arm or leg is left unburnt. A urethral catheter is inserted using a no-touch technique and measurements of urine output are noted every hour. This serves as the primary guide to fluid output. The catheter is used until intravenous fluids are stopped, and should be changed every 48 hours.

Fluid therapy must be accurately controlled.[15] This is not always an easy task, as the child's kidneys are relatively inefficient and cannot cope with wide variations of fluid exchange as can an adult's, and care is necessary to prevent overloading the circulation. The immediate need is for replacement of fluid, colloid and electrolyte that are lost through the altered permeability of the damaged skin.

The first fluid given must be plasma or serum. The common practice of running in saline first only loses valuable time and tends to aggravate the tendency to acidosis, as the extra chloride displaces bicarbonate[5]; colloid is what is needed. During the first 24 hours the amount of fluid needed is calculated according to a basic formula, a widely used one being that of Evans[7, 11]: 1 ml. per % area burnt per kg. body weight per 24 hours given as colloid plus 1 ml. per % per kg. given as electrolyte solution, plus basal requirements.

The variation on this formula most commonly used is 0.5 ml. colloid and 1.5 ml. electrolyte per 1% per kg. burnt. These estimates all apply to under 40% area burns as the calculation using larger figures will give an amount which is excessive and dangerous. Any burn greater than 40% surface area is estimated, therefore, as for a 40% burn area.

The Evans formula often tends to overload the circulation after the first 12-16 hours, as it is not easy to assess the potential oral intake which, in many children, is satisfactory during the vital first 24 hours. It has been found safer to base requirements on half that amount, i.e. 1 ml. per kg. per % area burn, giving half each of colloid and electrolyte and adding to this the daily basal fluid requirements according to age.

The volume of fluid to be given is best calculated on the basis of the first 48 hours' need, i.e. the modified Evans formula plus the basal requirement for 2 days and this total quantity is then divided into 3 equal parts.[10] The first part is given in the first 8 hours, the second in the next 16 hours, and the third quantity in the next 24 hours. In the first 24 hours, the first two portions of fluid are given intravenously, but during the second 24 hours, the

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third part is given partly intravenously and partly by mouth, depending on the amount of oral intake which will by then be established.

The amount given during the first 8 hours is the important factor, and this period must be estimated from the time the burn was sustained, and not from the time of admission. Usually this first 8 hours estimated amount will have to be given in 3 or 5 hours, depending on the time lag before admission.

Example: A child of 9 years weighing 25 kg. sustains a 30% burn of partial thickness loss.

Intravenous fluid required = 25 x 30 ml. = 750 ml. for 24 hours.
= 1500 ml. per 48 hrs.

Basal requirement for 48 *hours* = 3000 ml.

Total fluid required for 48 *hours* = 4500 ml. divided into 3 parts.
= 1500 ml. for each period.

In each of the first two periods 1500 ml. are given intravenously but in the second 24 hours this amount is given in part intravenously and the remainder orally.

While a formula helps as a basis, the speed and quantity of the intravenous infusion during the first few hours will vary. The urinary output should be maintained at 10-20 ml. per hour, in children under 1, and at 20-30 ml. per hour in older children; if it falls below this, the infusion should be speeded up by 100-300 ml. of 5% dextrose saline. If the blood pressure drops, the colloid solution should be rapidly infused. Restlessness is usually an indication for increased colloid rather than for sedation. Repeated haematocrit readings at 3- or 4-hourly intervals help determine the speed of infusion, which should be given rapidly until the packed cell volume is returned to near normal levels and the urinary output maintained at a satisfactory level. The remainder of the fluid requirement can then be given in accordance with the formula. Haematocrit estimations should be done by a simple method using heparinized 1.0 mm. bore glass tubes filled with capillary blood from finger, toe or ear, centrifuged and read against a standard scale. This enables repeated and rapid estimations to be made by the resident house surgeon at any time.

The variations in normal haematocrit and blood volume levels are indicated in Table 2.

Retention of sodium and water is usual during the first 4-5 days after burning[5] and this is similar to the retention occurring after any surgical operation. This retention is related to the extent of burn in that the higher intake needed in greater degrees of burns leads to early release of water on the third or fourth day. A high fluid intake in the early period

TABLE 2: APPROXIMATE NORMAL HAEMATOCRIT VALUES AND BLOOD VOLUME IN CHILDREN*

Age	*Haematocrit*	*Blood Volume* (Ml.)
Birth	68	200
1 month	45	250
3 months	38	300
1 year	38	500
3 years	38	1000
5 years	39	1300
7 years	40	1500
10 years	40	2000
Adult male	47	6000

(*After Gissane[10])

of a burn given in an attempt to maintain a normal or increased urinary output is not without some danger, and in the first 2-3 days the total amount of fluid calculated should rather err on the moderate side; Bull and England[5] confirm that this moderation does not prolong the oliguria or produce anuria or azotaemia.

Anuria has not been noted in burns in children in the series of 570 cases reported on, but two cases of anuria in adults have recently been seen where the initial haemoglobin reading within a few hours of burning was under 50%, and the haematocrit between 27 and 30, suggesting that severely anaemic patients who are burnt have such a low circulating blood volume that the added stress of the injury causes failure of renal function.

A moderate negative potassium balance develops during the first few days and tends to correct itself by the increase in intake of potassium. There appears to be no clear relationship of the potassium levels to the extent of the burn.[5]

The quantity and quality of electrolyte should be estimated carefully. Young children cannot cope with excess electrolyte as effectively as can adults, and therefore the total electrolyte solution calculated should be given in part as half strength Hartmann's solution, designed to provide disposable anions and cations, or as 5% dextrose in water.

Blood replacement in the early stages of burn treatment is essential. Despite a high initial haematocrit reading, bottled blood will, if anything, help dilute the concentration and will replace the not inconsiderable quantity destroyed by deep burns and help maintain an adequate Hb level. There has been controversy about the need for early blood infusion, but there is little doubt that in deep burns and all burns over 30% surface area, it is essential and often life-saving during the first two days.[18] Evans[7] has shown by blood volume studies that there is a decreased circulating red

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cell mass soon after a burn, and there is considerable clinical evidence to support its early use.[5, 7]

Drinking of nutritive fluids such as orange juice, glucose water and half strength saline, is encouraged as early as possible (1 oz. hourly or more). Children tolerate oral feeding well, and vomiting and ileus are uncommon. Fox's work[8] and that of investigators in Peru have indicated the value of giving saline by mouth. This corroborates Rosenthal's original observations[19] on burnt mice, where burn mortality was considerably reduced by subcutaneous saline; and, while not strictly comparable to the more complex picture in Man, oral saline would seem from available evidence to be of use.

Blood chemistry estimations should be done daily, but they are too crude to be relied on entirely in burns. Many patients die with a normal blood electrolyte picture. Unless the electrolytes are greatly disturbed, their variations will fall within normal limits, which will give a false picture of the clinical state. The clinical condition, urine output, haematocrit reading and blood pressure, in that order, are more reliable indications of progress.

The adrenal cortical response to the injury can be followed by total eosinophil counts and Sevitt[20, 23] indicates that the eosinophil response (40-200 eosinophils per ml. is the normal range) in severe burns give a reliable picture of adrenal cortical hyperactivity. Within a few hours an eosinopenia occurs, falling to low levels or to zero and this level continues for a number of days. In small burns under 10% a rise occurs within 2 days, whereas in severe burns the level only rises after 3-5 days. A more prolonged eosinopenia in a small burn suggests an under-estimation of the degree of shock and stress. A shorter period of eosinopenia than expected in a major burn indicates either a lesser degree of stress or evidence of adreno-cortical failure. A severe depression of eosinophils that continues beyond 6 days indicates a poor prognosis.

The daily eosinophil response should become a routine investigation in all serious burns; it may not always be accurate, but it does represent a help to rational therapy and serve as a prognostic guide.

Cortisone and ACTH have little place in the treatment of burn shock. Evidence both experimental[25] and clinical[18, 26] has accumulated to show that these drugs more often than not are dangerous in burn patients and may serve only to deplete an adrenal approaching exhaustion. There are no accurate indications for telling when adrenal cortical failure needs exogenous aid, and consequently cortisone is too often given as a desperate terminal measure. The advantages that ACTH is said to provide, such as supplementing depleted endogenous hormone in the severely burnt patient and providing a sense of well being in the later stages of treatment, are of doubtful value when weighed against the lessening of resistance to infection and the radically altered electrolyte balance.

By the second day the requirements must be re-assessed, and in this period the fluid is given partly intravenously (half as colloid and half as electrolyte solution) and the remainder orally, if the patient is drinking satisfactorily. Blood or plasma should form the colloid, and the electrolyte given as the remainder, intravenously in the form of half-strength Hartmann's solution.

The daily continuation of intravenous fluid is necessary as long as the loss by seepage continues, unless the oral intake can cope with both this and the daily metabolic requirements. Generally, intravenous fluids can be discontinued after 48 hours in most burns of 10-30% extent, provided daily blood chemistry and Hb estimations are checked and the clinical condition does not indicate dehydration. Daily accurate fluid balance estimation is continued, but loss from burn areas continues for some days, and often sodden bedclothes are seen despite crust formation.

Antihistamines, although not of proven value, should be given for the first three days.

LOCAL TREATMENT

Until shock is controlled, and this may take up to 24 hours or even longer, no local treatment is undertaken other than sprinkling penicillin powder on to exposed areas. Whilst this may appear contradictory to the principle of preventing exudation, it is essential for the welfare of the patient that he is disturbed as little as possible. When the acute phase of shock is passed, with the blood pressure normal and the fluid requirement stabilized, local treatment is undertaken under pre-medication, or a light anaesthetic in the ward, with strict asepsis. Blisters are snipped and pressure dressings consisting of penicillin cream on gauze, liberal quantities of cotton wool and crepe bandage are firmly applied to limbs.

There has been controversy concerning the relative efficiency of the exposure method used

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by Wallace in particular and the method of occlusive dressings.[18] Either method properly applied is satisfactory. Both in fact are 'closed' methods, and both have advantages and disadvantages: a combination of both is a suitable compromise. Pressure dressings are simple to apply to limbs, comfortable for the patient, and would seem to have the advantage of allowing some movement without maceration. On the other hand, these dressings are difficult to apply and maintain on burns of the perineum, abdomen, chest, back, neck and face, soon work loose and become uncomfortable. For these the encouragement of crust formation by the open method is advised. Such areas must be constantly attended to and, where the crusts crack, further applications of penicillin powder applied. When the back is burnt the patient is laid on sterile gamgee covered with penicillin powder. In a sense this, too, is a form of pressure dressing. In hot climates extensive pressure dressings induce hyperpyrexia and, as the normal physiological response by sweating is reduced by skin destruction, patients tend to pant and hyperventilate to increase the respiratory fluid output.

During the next few days, constant nursing care is necessary and attention paid to the position of limbs. Elevation of both lower and upper limbs helps to diminish the burn oedema, which is often marked.

Burns of the neck tend to macerate because of the fatty rolls, and a soft pad should be placed under the shoulders to hyper-extend the neck and help crust formation in the neck flexion creases. The arms should be abducted with the elbows partly extended and the hands rested on pillows to avoid sogginess in the axillae and elbow creases.

By the eighth day dressings, if any, or crusts should be removed, and this procedure can be rendered relatively painless by strong analgesic premedication and soaking the dressings off in a warm bath. The care with which treatment is performed at this time will determine the child's behaviour on future occasions.

A bacteriological swab is taken and careful re-assessment of the extent and depth of the burn is now made. Often it is found that the initial assessment of the burn was incorrect and for the first time an accurate picture of full thickness loss may be possible. The haemoglobin level is again checked and blood infusions of 100-300 ml. given at intervals to correct any deficit. The haemoglobin should not be allowed to fall below 70%, and preferably kept nearer 80%, the normal level for children. Where possible, within 12-14 days, initial grafting procedures should be undertaken, preceded by appropriate antibiotics.

ANTIBIOTICS AND BACTERIOLOGY

Burns are sterile for a short time after the injury. Within 12 hours of admission to hospital mixed organisms can be cultured, and it is essential that a bacteriological swab be taken immediately on admission as a control. Most of the organisms are initially penicillin-sensitive, and penicillin 250,000 units should be given intramuscularly every 6 hours. Colebrook has cast some doubt on the use of parenteral penicillin and suggests relying on local administration, while Wallace relies on the parenteral penicillin alone to keep the exudate free of organisms. However, it is preferable that both methods be used. With strict attention to asepsis, many burns can be kept free of organisms. Different varieties of streptococci and staphylococci are commonly found after the first dressing, and these must be tested for sensitivity to specific antibiotics. *B. proteus* and *B. pyocyaneus* are dreaded organisms, usually remaining on the surface and preventing successful grafting. The value of local Polymixin (0.1 mg. per g. in a Lanette wax base) has been proven by Jackson[13] for these organisms with parenteral treatment given as well. The haemolytic streptococcus is the most serious organism encountered, may cause a fatal septicaemia, and precludes any attempt at grafting. Sensitivity to broad-spectrum tetracyclines is usually necessary for staphylococci as they may often become penicillin-resistant. Constant bacteriological control of organisms is needed before and after each operation, particularly where there has been only a partial take of skin grafts, as this is the only method of ensuring rapid control of infection.

The presence of pus due to mixed organisms does not always contra-indicate grafting and, depending on the type of organism and the sensitivity to antibiotics, skin coverage should not be delayed in special areas.

PRINCIPLES OF SKIN REPLACEMENT

Waiting for burn slough to separate naturally is contrary to the principles of treatment. The dead skin should be excised as soon as the patient is judged fit for such an operation. If

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left, the slough increases toxic absorption, makes further demands on the body's resources for dealing with dead tissue and, as important, for dealing with dead tissue and, as important, increases the amount of fibrosis.

The excision of burn slough is a major procedure, and is invariably accompanied by a considerable blood loss. It is imperative for a blood infusion to be running before the procedure starts so that the blood loss can be anticipated. Unless this is done, the anaesthetist may have considerable difficulty in resuscitating the child. No more than 4-5% of replacement by autogenous skin graft is possible at each stage, as a 5% grafting is equivalent, together with the donor site, to a 10% skin loss during the operation.

Grafts are cut by hand or by an electric dermatome and applied on tulle gras as strips or as stamp grafts, with care to place the long axis of the strips along tension lines.

Following the grafting, both recipient and donor areas should have firm pressure dressings applied. When the abdomen, back, perineum or neck are grafted, they require firm pressure to help the graft take. Most children will manage to wriggle out of almost any dressing around the head and neck, so great care is necessary in applying it as firmly as possible, and a light plaster shell completes the dressing. Transparent tape has been used on the neck to keep the graft in place, but it seems to have little advantage over carefully applied pressure dressings.

In cases when multiple grafting is necessary, the skin replacement of digits and flexor surfaces of elbow, popiteal fossa, groins, axilla and neck must take precedence.

Where large areas need grafting, homografts are valuable and may on occasion be life-saving.[14] These homografts will act as a primary dressing and so provide a cover even though they separate and die after 14-21 days. The chief indication is the lack of sufficient of the patients' own skin, and in burns over 30-40% the need for skin cover is vital to increase the chance of survival and to preserve function. When autografts are limited, they are used for priority areas such as fingers and flexion creases, while homografts are used to cover the less important areas. Less frequently they are used as a means of skin cover in patients too ill from associated pneumonia or toxaemia to allow of an anaesthetic.[14]

Recently[2] post-mortem homografts have been used with success and herein lies a practical method in large burn centres. The time of survival of the stored skin is not known.

Homografts should be laid in strips alternating with strips of autograft skin so that the latter will help supply an outgrowth of epithelium when the homograft is shed.

When excess skin has been cut during a grafting operation, the extra pieces should be kept in saline-soaked gauze in a refrigerated sterile tin. It can be used up to 4 weeks later as a homograft.

The grafted areas are inspected on the fifth or sixth day, preceded by premedication, and the dressings carefully soaked off in a bath. Skin which has taken will be firmly adherent by this time, and the layers which tend to peel off with the dressings are often cuticle only. Where patches of donor skin have not taken, there will usually be a thin layer of pus lying on sprouting granulation tissue. A bacteriological swab is again taken and firm dry dressings applied to the area. Haemoglobin is repeatedly checked and, where necessary, small blood infusions given and further grafting proceeded with as soon as possible.

Areas of deep partial thickness loss are the most difficult to assess. Many of these, by the 14th to 18th day, will have been completely covered by skin but where, at the first operation, there are doubtful areas where even minimal scarring would be detrimental, it is safer to graft these as well. If the child's own skin regenerates, the grafts will simply be shed and will, in any case, have acted as an excellent dressing.

Even where there has been a 90% success following grafting, small irregular islands of granulation are often left. If these are larger than 2 cm. in diameter, a little more than the area covered by a sixpence, they should be re-grafted after scraping the granulations flat. This will lead to much quicker healing than allowing the area to epithelialize on its own.

Despite good primary 'takes' in areas behind the knees, in the elbows or axilla, great care must be taken not to allow excess movement here for some weeks. The limbs should be immobilized in a bivalved plaster shell and gentle daily movements carried out by a physiotherapist, otherwise the new epithelium will split and probably lift off from the increased tension of fibrosis and contraction. Tension is an important factor in late failures of grafting. In the case of the neck and flexion creases, despite early grafting, contractures may rapidly form, sometimes within 2-3 weeks. These should be excised immediately and widely, together with the underlying

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fibrous base, and split skin grafts applied. Despite this there may be further contractures, in which case it is best to allow the child home for re-admission after 3-4 months for further correction of the fully contracted scar, when active fibrosis has ceased. As the child continues to grow, the reconstructed area may require further plastic procedures.

Fingers are a particularly difficult problem as full thickness loss over the dorsal aspect so often opens inter-phalangeal joints. Such fingers should be grafted as early as possible, and immobilized in the position of function. Despite this, fingers are often rendered stiff and useless and may require amputation.

NUTRITION

During these weeks the child must have a high protein diet supplemented by protein hydrolysates and, where necessary, serum proteins must be estimated.[4]

A particularly unfortunate feature sometimes seen in non-White patients is the late sloughing of both recipient and donor sites. On a few occasions patients have been discharged from hospital with fully healed burn and donor sites, only to be re-admitted with both areas denuded in patches with full thickness skin loss. In two children so admitted, despite repeated grafting to the original donor site, most of the area ultimately healed by fibrosis. Some relationship of this misfortune to a chronic malnutritional state is assumed, but this will need further investigation.

Another disheartening feature is the occasional occurrence of complete grafting failures despite careful bacteriological and haemoglobin checks. Yet, for no discernible reason, grafting after some weeks is sometimes suddenly successfull with no apparent relationship to the organisms which are present. This too, might be related to the general nutritional state of the patient. Since protein loss is a major feature of a burn both in the shock phase and later following pus formation, serum protein levels have been taken as a guide. Many of these patients have a low albumin level often below 1 g. %. High protein diets have been arranged and protein hydrolysates given intravenously and by mouth; yet chronic malnutritional states are so complex and result in such widespread derangements[9] that concentration on one aspect only of this problem may be of little help, perhaps precipitating other deficiencies at the same time. Proteins, although probably of help in the acute stages of the injury, are of less value in overcoming the long-term effects of years of poor nutrition upon which the success or otherwise of the reparative surgery depends.

SUMMARY

The methods of assessment of the extent of burns is described. Accurate assessment is the necessary basis for the treatment of burn shock.

The control of fluid balance is discussed in detail and the need for blood infusion in the early stages in stressed.

Shock is still the biggest factor in burn deaths, and fluid replacement is as urgently required as in cases of haemorrhage. It requires constant supervision for the first 12-16 hours. The best guides to the patient's progress are the clinical state and the hourly urine secretion measured by an indwelling catheter.

Infection, the second commonest cause of death, and the cause for much subsequent reparative surgery, must be reduced to a minimum by treating burns in the same strict fashion as any open wound is treated under operating theatre conditions.

The local treatment is a matter of choice between the two accepted methods, but experience and preference determine what is necessary.

Repeated bacteriological control is essential and the haemoglobin level must be constantly checked.

Early grafting is necessary to preserve function and save life and homografting is a valuable aid in extensive burns.

Chronic malnutrition is a possible factor related to early and late failures of grafting.

There are still many unexplained features of burns, particularly those concerning fluid and electrolyte distribution, but vigorous anti-shock measures are now succeeding in keeping many severely burned patients alive for much longer periods than before. Whilst these patients may still die of other complications, some of the answers to this aspect of burn shock are becoming clarified.

The treatment of burns belongs to the broad field of general surgery. The provision of a special unit is a rational method of centralizing the care of burn patients. These injuries represent an example of the complexity of a disease process where problems of organization, shock treatment and fluid therapy, control of infection, nutrition and restoration of function by surgical means are all concerned.

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OPSOMMING

Die metodes vir die vasstelling van die omvang van brandwonde word beskryf. Akkurate vasstelling is die onontbeerlike basis vir die behandeling van die skok wat op verbranding volg.

Die beheer van die vloeistofewewig word breedvoerig bespreek, en die noodsaaklikheid van bloedinfusie in die vroeë stadiums word beklemtoon.

Skok is nog steeds die belangrikste bydraende faktor tot die sterfgevalle wat op verbranding volg, en vervanging van die vloeistof is net so noodsaaklik as in gevalle van bloeding. Gedurende die eerste 12-16 uur is gedurige toesig noodsaaklik. Die beste aanduiding van die vordering wat die pasiënt maak, is sy kliniese toestand en die urine-afskeiding iedere uur, soos gemeet deur 'n ingesteekte kateter.

Infeksie, die tweede belangrikste oorsaak van die dood, en die rede vir veel van die herstellingschirurgie wat later onderneem moet word, moet tot 'n minimum verminder word deur die brandwonde net so sorgvuldig te behandel soos enige oop wond onder operasiesaaltoestande.

Die plaaslike behandeling bied die geneesheer 'n keuse tussen die twee aanvaarde metodes, maar ondervinding en voorkeur bepaal wat nodig is.

Herhaalde bakteriologiese beheer is noodsaaklik, en die hemoglobienpeil moet gedurig gekontroleer word.

Vroeë inplanting is noodsaaklik om die funksies in stand te hou en 'n lewe te red, en homoinplanting is 'n waardevolle hulpmiddel in gevalle van uitgebreide brandwonde.

Een van die faktore wat bydra tot die mislukking van vroeë en laat inplantings is bes moontlik chroniese ondervoeding.

Daar is nog steeds talle onverklaarbare kenmerke van brandwonde, veral dié wat betrekking het op vloeistof- en elektrolitiese distribusie, maar kragdadige skokbestrydingsmaatreëls slaag tans daarin om talle erg verbrande pasiënte oor veel langer tydperke as ooit tevore aan die lewe te hou. Terwyl hierdie pasiënte nog steeds ten gevolge van ander verwikkelinge kan sterf, het sommige antwoorde op hierdie aspek van brandwondskok nou duidelik begin word.

Die behandeling van brandwonde ressorteer onder die breë gebied van algemene chirurgie. Die beskikbaarstelling van 'n spesiale eenheid is 'n rasionele metode om die behandeling van pasiënte wat aan brandwonde ly, te sentraliseer. Hierdie beserings is 'n voorbeeld van die ingewikkeldheid van 'n siektetoestand waarby nie alleen organisasieprobleme betrokke is nie, maar ook die behandeling van skok, vloeistofterapie, die beheer van infeksie, voeding, en die herstel van funksie deur middel van chirurgie.

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BURNS AND SCALDS

PROBLEMS IN THEIR TREATMENT

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This paper contains the clinical impressions of 17 years of active practice and interest in the management of patients with burn and scald injuries. There is considerable divergence of opinion about how these economically important injuries are best treated, and consideration of the medical problems of nuclear warfare reveals the sombre fact that survivors will

inevitably suffer from varying grades of this type of trauma.

1. INITIAL GENERAL TREATMENT

(*a*) PROBLEMS OF INTRAVENOUS THERAPY

Burning or scalding of the surface of the body is accompanied by considerable changes in the intracellular and extracellular fluids, and the local and the general circulation fluids apart from more drastic physico-chemical changes in the involved tissues themselves. A protein-rich exudate flows out of the local capillary bed into the injured tissues. The rate of this outflow follows a parabolic curve for the first 48 hours and the local and general reactions of the body lead to re-absorption, beginning about 72 hours after the injury. This is undoubtedly true for the superficial burn, but in the deep burn only the fluid lost into the periphery of the dead and potentially dead tissue is available for re-absorption; also, in these cases, the blood and tissue fluid trapped, by burning, in the dead tissue is irrevocably lost to the bodily economy—and this is important in extensive deep burns. Re-absorption can take place only through comparatively normal capillary and lymphatic channels and, no doubt, part of the reason for the time lag in re-absorption is associated with the re-establishment of this absorptive system. In deep burns there is probably a further flow, in the adjacent tissues, of blood and tissue fluid as a response to the irritation of the living tissue from the continuity with dead tissue, i.e. all the phenomena of inflammation are called into play to initiate a line of demarcation.

Burns and scalds of any dimension (an arbitrary rule is over 9% surface area in children and over 15% in adults) are usually accompanied by 'shock', with phenomena of loss of circulating blood, etc. from the bodily economy. In short, the big problems of the early stages of burns are those of local and general tissue anoxia, and renal insufficiency with its attendant loss of control of electrolytes, metabolites, organic waste, water, etc. in the circulation.

Shock is still the commonest cause of death in burns; in contrast to other causes of shock there is often a latent period of about 2 hours before clinical features of shock are apparent in burns cases.

To summarize, body fluids may be lost as follows:

1. Exudation into the burnt area and the adjacent tissues.
2. Irrevocable blood loss due to coagulation of blood in the vascular bed of damaged tissues.
3. General loss of blood fluids due to shock mechanism.

(Later the following factors can come into play:

4. Delayed haemolysis of damaged red cells.
5. 'Post-traumatic anaemia.'
6. Depression of bone marrow tissues by already existing anaemia, toxaemic products from tissue and sepsis, hypoproteinaemia and depressed food and fluid intake).

Problems of Acute Renal Insufficiency. This condition may be defined as a hyperacute, but *reversible,* loss of renal function due to renal ischaemia (and anoxia) and subsequent diffuse areas of tubular degeneration. Even in those patients who do not present with customary features of shock, e.g. peripheral vascular collapse, there is often a deficient circulating blood volume. Experiments have shown that the renal vascular bed is affected to a very great degree in the vasoconstrictor responses to shock. The renal ischaemia is said to be proportionately greater than the reduction in cardiac output and the glomerular filtration rate and tubular excretion function are greatly reduced. Renal vasoconstriction and reduction of glomerular filtration, together with back diffusion of the filtrate due to damage to the tubular cells, accounts for the oliguria.

Water and Salt Imbalance. From available evidence it appears that much of the post-traumatic disturbance of water and salt metabolism is due to:

1. Secretion of anti-diuretic hormone.
2. Haemodynamic changes in the kidneys.
3. Adrenocortical activity.

In normal circumstances the kidneys have an enormous efficiency and can easily excrete excesses of salt and water; but in post-traumatic conditions this efficiency is markedly impaired and, if oligaemia of shock also occurs, then this impairment becomes a very grave complication.

Potassium Imbalance. In burns, potassium is lost from the injured tissue cells and this is released into the extracellular circulation. If there is impairment of renal function, this potassium may accumulate in the extracellular fluid and reach a toxic level. When the renal function is normal, potassium liberated from cells is progressively excreted.

Electrocardiographic Changes in Potassium Abnormalities. This type of investigation can be helpful in the detection of gross potassium imbalance.

(*a*) *Features of Low Potassium Levels*:

1. The QT interval is prolonged.
2. The RST segment is depressed.

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3. The T wave is lowered, flattened and prolonged.

(*b*) *Features of High Potassium Levels*:

1. The PR interval is prolonged.
2. The P wave may disappear.
3. The QRS complex is prolonged.
4. The T wave increases in amplitude and becomes peaked; eventually there is intraventricular block.

From a broad consideration of all the known and suspected results of trauma on bodily economy, e.g. fluid and electrolyte imbalance in cells, in the extracellular fluid and in the blood circulation fluid, hormonal imbalance, upsets in nitrogen, carbohydrate and fat metabolism, etc. the following facts seem to emerge:

1. Estimation of the circulating blood content of potassium, sodium, chlorides, bicarbonate, protein ratio, water, cell : plasma ratio, etc. do not reflect accurately the *status quo ante* in the cells and the interstitial fluid circulation. Therefore frantic attempts by intravenous therapy to restore a low reading of any of these constituents to an alleged average normal reading can be dangerous.

2. Certain alterations in ionic values are normally part of the body's reaction to the stress of trauma and should not be altered indiscriminately by intravenous therapy.

3. The natural physiological controls should be brought into play as soon as possible, e.g. the ingress via the alimentary canal of a normal type of diet and fluid intake, and the selective excretion function of normally working kidneys.

4. Kidneys can function correctly if there is no general or local renal anoxia, and a continuous and adequate renal blood supply is available.

5. In these modern days one is apt to forget that the body has a great capacity of coping with the effects of trauma, and the clinician should approach the problem in the humble capacity of an inferior servant to Nature and not as an omniscient scientist with an intravenous therapeutic wand.

6. The clinician must ensure that the body has the wherewithal to function adequately. Chiefly, this is a satisfactory blood volume to obviate anoxia—the greatest enemy to recovery. He must make calculated estimations of fluid and electrolytes necessary to tide the patient over his initial crisis, and he should institute feeding by mouth as soon as possible.

It will be appreciated from the foregoing discussion that anoxia from oligaemia of shock must be prevented by whole blood transfusion given as soon as possible; and water and electrolyte replacement by the minimal necessary intravenous transfusion given over the 24 hours succeeding the injury.

Oral feeding of fluids and a mixed light diet should be instituted as soon as possible. Where the patient is reluctant to take oral feeds, feeding can be done through a soft nasal gastric tube. In very severe burns there may be anything from mild gastric reflex dysfunction to established acute dilatation of the stomach. This will introduce further problems of fluid and electrolyte loss from the bodily economy and inability to start early oral feeding. This complication necessitates continuation of intravenous fluids for the minimal possible time, very accurate fluid intake and output charts, hourly charting of urine and maintenance of calorie needs with invert sugar, alcohol, etc.

How Much, and What Type of Intravenous Fluid should be Given? The following physiological facts have a bearing on this subject.

The maximum expansion of the extracellular fluid space compatible with safety is about 50%, i.e. about 10% of the body weight.

In an adult weighing 70 kilograms:

The *Total Body Water* is 60% body weight, average 42,000 c.c.

The *Intracellular Water* is 38.5% body weight, average 27,000 c.c.

The *Extracellular Water* is 21.5% body weight, average 15,000 c.c.

(*a*) *Interstitial Water* is 16.5% body weight, average 11,500 c.c.

(*b*) *Blood Water* is 5% body weight, average 3,500 c.c.

Therefore, for practical purposes, the maximum expansion by intravenous fluids, with any safety, is in the neighbourhood of 7 litres; and this presupposes resilient cardio-vascular and renal systems. Too little fluid will tend towards perpetuation of oligaemic problems and too much will lead to water intoxication, pulmonary oedema, etc. The margin of safety is very small.

In babies and children the controlling power of the kidneys over water and electrolytes is still not completely developed and there is virtually no margin of safety.

The Invert Sugar—Alcohol (Baxter) solutions are a very satisfactory source of essential calories, but the intravenous amino acid replacement fluid is of little assistance to the patient during the first 4 days of the post-traumatic period.

GUIDANCE NOTES CONCERNING USE OF THE PYROGRAM COMPUTATIONS (TABLE 1)

1. All patients must receive total (A) by mouth as glucose water each 24 hours.

2. All patients suffering from superficial burns of less extent than 9%, and who never manifest any feature of shock at any time, may have total fluid in (B) plus total fluid in (A), and the whole taken *per os* as glucose water.

3. For superficial burns of 9-20% and for any patient with a less than 9% burn who manifests any feature of shock, give the total

TABLE 1: FLUID REPLACEMENT

DETAILS DRAWN FROM THE PYROGRAM READY RECKONER (ALLEN & HANBURY)

Age (years)	(A) *Fluid by Mouth* (*in c.c.*) (All cases)	(B) *Fluid by the Intravenous Route* (*in c.c.*) Percentage Area of Body Burned														
		2%	4%	6%	8%	10%	12%	14%	16%	18%	20%	22%	24%	26%	28%	30%
Birth—3/12	700	20	40	60	80	100	120	140	160	180	200	220	240	260	280	333
3/12—6/12	900	28	56	86	112	140	168	196	224	252	280	308	336	364	392	466
6/12—9/12	950	32	64	96	128	160	192	224	256	288	320	352	384	416	448	600
9/12—1	1000	36	72	108	144	180	216	252	288	324	360	396	432	468	504	666
1—2	1050	40	80	120	160	200	240	280	320	360	400	440	480	520	560	866
2—3	1100	56	112	168	224	280	336	392	448	504	560	616	672	728	784	1000
3—4	1150	64	128	192	256	320	384	448	512	576	640	704	768	832	896	1133
4—5	1200	72	144	216	288	360	432	504	576	648	720	792	864	936	1008	1200
5—6	1250	84	168	252	336	420	504	588	672	756	840	924	1008	1092	1176	1333
6—7	1300	92	184	276	368	460	552	644	736	828	920	101	1104	1196	1288	1466
7—8	1350	108	216	324	432	540	648	756	864	972	1080	1190	1296	1408	1512	1600
8—9	1400	116	232	348	464	580	696	812	928	1044	1160	1276	1392	1508	1624	1733
9—10	1540	124	248	372	496	620	744	868	992	1116	1240	1364	1488	1612	1736	1933
10—11	1600	136	272	408	544	680	816	953	1008	1224	1360	1496	1632	1768	1904	2133
11—12	1800	148	296	444	592	740	888	1036	1184	1332	1480	1628	1776	1924	2072	2333
12—13	2000	164	328	492	656	820	984	1148	1312	1486	1640	1804	1968	2132	2296	2600
13—14	2200	180	360	540	720	900	1080	1260	1440	1620	1800	1980	2160	2341	2520	2866
14—15	2400	208	416	624	832	1040	1248	1456	1664	1872	2080	2288	2496	2704	2910	3200
15—16	2600	236	472	708	944	1180	1416	1652	1888	2024	2360	2496	2832	2968	3304	3600
16—17	2800	256	512	768	1024	1280	1536	1792	2048	2304	2560	2816	3072	3328	3584	3933
17—18	3000	280	560	840	1120	1400	1680	1960	2240	2520	2800	3080	3360	3640	3920	4333
Adult	3000	300	600	900	1200	1500	1800	2100	2400	2700	3000	3300	3600	3900	4200	4666

in (B) intravenously as follows:

Half as plasma, and half as Compelitre Fluid (Baxter) for adults;

Half as 5% Invert Sugar in 0.2% Sodium Chloride for children.

4. For deep burns of any dimension and all superficial burns over 20% extent, give total (B) intravenously as follows:

Half as fresh compatible blood, quarter as plasma and the last quarter as the electrolyte fluids mentioned in (3).

5. As a general rule, give blood or plasma before giving electrolyte fluids.

Give 50% of total fluid in (B) during the first 8 hours and the remaining 50% over the subsequent 16 hours.

6. If for any clinical reason the patient's general condition suggests that the above therapy has been inadequate, give half the (B) total as similar fluids over the next 24 hours.

7. If after 24 hours, the urinary output is unsatisfactory and the figures recorded are below the lesser normal figure, as shown in the *Table of Urinary Output* (Table 2), give a maintenance dose, by mouth or by slow drip per 24 hours, of 5% Invert Sugar in water, equivalent to one third of the fluid computation shown in (A), until diuresis is established.

Probably the easiest method of estimating the percentage of surface burnt is the *Rule of Nines* (Wallace):

Whole head—9%.
Each upper limb—9%.
Front of trunk—18%.
Back of trunk—18%.
Each lower limb—18%.
Perineum—1%.

As the clinician is often apt to overestimate the area involved, it is a good plan to do an independent estimate of the non-burnt area, add the two results together and check if they are complementary.

THE IMPORTANCE OF URINE OUTPUT

Without doubt, the amount and character of the urine excreted is one of the best guides to the success of any intravenous therapy, because it reflects accurately the state of the internal fluid balance, more accurately and long before the usual laboratory examinations do, e.g. blood biochemical estimations, haematocrit readings, etc. Notwithstanding this, the urine results must be considered in conjunction with

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TABLE 2: TABLE OF URINARY OUTPUT

DETAILS FROM THE PYROGRAM READY RECKONER (ALLEN AND HANBURY).

Age	*Weight*		*Satisfactory Hourly Urine Output in C.c.*
	Lb.	*Kg.*	
Birth—3/12	11	5	8—20
3/12—6/12	15	7	8—20
6/12—9/12	20	9	8—20
9/12—1	22	10	8—20
1—2	29	13	20—24
2—3	33	15	20—24
4—5	40	18	20—24
5—6	44	20	24—28
7—8	53	24	24—28
8—9	57	26	24—28
9—10	64	29	30—35
10—11	70	32	30—35
11—12	77	35	30—35
12—13	86	39	35—50
13—14	95	43	40—60
14—15	106	48	50—100
15—16	119	54	50—100
16—17	130	59	50—100
17—18	141	64	50—100
Adult	154+	70+	50—100

general clinical signs, e.g. an obviously dry coated tongue, inelastic skin, soft rapid pulse, increased respirations, the restless apathy, etc. 'Burns should be treated by clinicians, not scientists'.

It should, however, be remembered that a transient oliguria may be a post-traumatic response, may persist for 72 hours or longer and may then be followed by a diuresis with urinary excretion of electrolytes. The approach to this complication is critically to review the whole management of the case and, if everything indicated has been done, a policy of 'hands off' is indicated during the oliguria, replacing only such fluid as is lost from the body.

Fluids containing potassium and sodium chloride should not be used until adequate passage of urine begins, with not more than 1,000 c.c. of 5% Travert (Invert Sugar) in water per 24 hours for the adult. Above all, the clinician must resist the temptation to administer quantities of glucose in water, or some fluid of this type, in dangerous attempts to force a diuresis.

It is advisable to pass a catheter soon after admission to hospital and to measure the urine collected every hour until the patient is well out of danger clinically. Very young patients, seriously ill patients and nervous patients can be better assessed in this manner.

The minimum normal excretion of urine in an adult is about 500 c.c. and, in these circumstances, the Sp. Gr. of the urine will be about 1030. In normal circumstances the Sp. Gr. of urine is usually between 1012 and 1020, and readings below 1010 suggest that too much fluid is being given. Therefore, instructions should be given to the nursing staff to collect and measure the hourly urinary output and to note the figure in c.c. on a special chart. At 4-hourly intervals the Sp. Gr. of urine collected during that period should be measured and the result appropriately charted.

2. DRUGS WHICH ARE HELPFUL IN THE TREATMENT OF THE SHOCK, ETC. OCCURRING IN BURNS

(*a*) *ACTH and Cortisone Derivatives.* Clinical experience has suggested that patients may be benefited by administration of ACTH or cortisone products during stress following severe burns; but a critical survey of the literature on this point reveals the following:

1. The mortality is *not* decreased by these drugs, but it is possible that borderline cases may be carried successfully through the period of stress.
2. There is a clinically noteworthy decrease in pain in cases so treated.
3. Control of fluid and electrolytes seems easier.
4. The presence of cortisone derivatives, given intravenously, enhances the good effects of noradrenaline in the treatment of shock.
5. There is a significant improvement in appetite, and a reduction in nausea, vomiting and apprehension in patients so treated.

If hydrocortisone is used, the writer gives it intravenously using Solu-Cortef (Upjohn & Co.). In desperate cases 100 mg. dissolved in 2 c.c. of sterile water is injected into a vein, taking 60 seconds for the injection. In other cases the drug can be added to the Vacoliter for sustained drip delivery.

In severe surgical stress the output of the normal suprarenal cortex is estimated to exceed 100 mg. of hydrocortisone during the first day, and over the succeeding 4-8 days there is a gradual falling off and a gradual return to normal output (20-25 mg. per 24 hours). Therefore subsequent dosage must be adapted to this information and the particular patient's requirements.

(*b*) *Noradrenaline* (*Levophed - Winthrop*). This drug functions as an overall vasoconstrictor without change, or only slight decrease, in the cardiac output. It is indicated for maintenance of the blood pressure in acute hypotensive states resulting from surgical trauma,

acute blood loss and profound shock from burn injuries.

Manner of Use and Dosage. Add 4 c.c. of Levophed 1 : 1,000 solution to 1,000 c.c. of isotonic sodium chloride solution, of 5% Invert Sugar solution, of plasma, etc. Each 1 c.c. of this dilution contains 4 micrograms of Levophed (as bitartrate). Give this dilution intravenously through a previously calibrated Murphy drip bulb permitting an accurate estimation of the rate of flow in drops per minute. After observation of the response to an initial dose of from 0.25 to 0.5 c.c. of dilution per 10 kg. of body weight per minute, adjust the rate of flow to produce and maintain the desired level of blood pressure. Although there are individual variations, the average dose to maintain the desired level of blood pressure ranges from 2 to 4 micrograms (0.5 to 1 c.c. of the dilution) per minute.

Caution. Because of its potency and because of the varying response to pressor substances, the possibility always exists of producing dangerously high blood pressure with overdoses of Levophed. It is desirable, therefore, to check the blood pressure every 2 minutes from the time the drug is started until the desired blood pressure is obtained; then every 5 minutes if the administration is to be continued. The rate of flow of Levophed must be watched constantly, and the patient should never be left unattended while receiving this drug.

The noradrenaline drip should be stopped 10 minutes after the desired blood pressure readings have been recorded and further readings taken every 5 minutes to ensure that they are maintained. Falling off of the pressures indicates the need to start the drip again.

Recent clinical evidence suggests that concomitant hydrocortisone intravenously enhances greatly the beneficial effects of noradrenaline; and the writer feels that patients who require these serious anti-hypotensive therapies also require intra-arterial blood transfusion—for this régime seems to be more successful than the combination of these drugs plus the conventional intravenous blood transfusion.

(*c*) *Methedrine.* Methedrine brand Methylamphetamine Hydrochloride is a derivative of amphetamine with distinctive therapeutic properties. Injected parenterally, Methedrine produces a prolonged rise of blood pressure by peripheral vasoconstriction, and an increase in pulse rate and pulse pressure. In addition to its vascular action, the drug is a very powerful central stimulant and increases the rate and depth of respiration.

Indications. Extensive clinical experience has established Methedrine as an efficient, dependable pressor agent with a prompt and sustained action in circulatory depression or impending shock during or following surgical anaesthesia. Methedrine has also proved a valuable adjuvant to resuscitative measures in the treatment of established shock, vasomotor collapse and narcotic poisoning.

In cases of established shock accompanied by severe reduction of blood volume, the value of Methedrine alone, like that of other pressor drugs, is doubtful. *Fluid replacement, adequate both in quality and quantity,* must be the first essential measure, but it may be supplemented with advantage by the injection of Methedrine.

For a single injection the usual dose range is 15-30 mg. (0.75-1.5 c.c.) intramuscularly, or 10-20 mg. (0.5-1.0 c.c.) intravenously. Table 3 serves as a rough guide.

3. RELIEF OF PAIN

The usual pain relieving drugs are probably not completely effective and depress the patient's general activity as well as his vital functions tending, for example, to chest complications. In the case of young children it is difficult to choose a suitable analgesic.

Pain in burns is curious in that it seems to be intense initially but less so after about 72 hours. I have therefore tried out a scheme of intravenous Procaine analgesia. (This therapy, as an adjuvant during major operations, impressed me greatly when I was in the United Kingdom, with particular reference to the long duration of freedom from post-operative pain in cases where this was usually a prominent feature).

TABLE 3

Condition	*Dose and Route* (I.M. = Intramuscular) (I.V. = Intravenous)
Surgical hypotension. Systolic blood pressure below:	
90 mm. Hg.	15—20 mg. (0·75—1·0 c.c.) i.m.
80 mm. Hg.	25 mg. (1·25 c.c.) i.m.
70 mm. Hg.	25—30 mg. (1·25—1·5 c.c.) i.m; or 10—15 mg. (0·5—0·75 c.c.) i.v. and 15—20 mg. (0·75—1·0 c.c.) i.m.
60 mm. Hg.	15—20 mg. (0·75—1·0 c.c.) i.v. and 15—20 mg. (0·75—1·0 c.c.) i.m.

The details of this analgesic therapy, which must be of brief duration, etc. bearing in mind the foregoing remarks on electrolyte intravenous therapy, are as follows:

Action of Procaine. This is mainly directly on terminal nerve endings. After about 10 seconds the patient feels a pleasant generalized warmth, commonly there is a feeling of inebriation tending to light-headedness. There may be a slight increase in blood pressure and respiratory depth and frequency. There is evidence that Procaine promotes urinary excretion.

Method of Administration. Give a barbiturate, e.g. intramuscular injection of phenobarbitone:

Up to or 2 years, gr. 1;
2-5 years, gr. 1½;
5-10 years, gr. 2;
Adults, gr. 3+;
On admission to hospital (see treatment of *Toxic Effects* below).

Dissolve 1 gr. of Procaine in 1,000 c.c. of Compelitre or 5% Invert Sugar in water (e.g. 1% solution of Procaine) and administer at about 40 drops per minute until suitable analgesia is obtained.

Procaine is hydrolysed very quickly by procaine-esterase to para-amino-benzoic acid and di-ethyl amino-etanol, which are excreted in the urine.

Cleaning, etc. of the burns can be started very soon after beginning the drip, if the patient's condition permits.

The duration of analgesia is difficult to predict, but I have seen it last for 30 hours in 4 cases—and it is always more effective if used early in treatment.

Advice is not available for dosage in small children, but it would seem that 50 c.c. of the solution given at 20 drops per minute would be effective and safe for older children and half this quantity for very small children.

Toxic Effects: *Symptoms.* These are:

1. Syncope, dizziness, headache, apprehension.

2. Dyspnoea, palpitations, precordial pain, nausea or vomiting.

Signs. These are:

1. Pallor, sweating.
2. Excitement or convulsions.
3. Fall in blood pressure—rapid pulse, air hunger.

Treatment: (*a*) *Prophylactic.*

1. The preliminary barbiturate lessens the possibility of the toxic effects.

2. Avoid the therapy in very old patients with severe arteriosclerosis.

(*b*) *Active.*

1. Stop the drip and lower the head.

2. Ventilate with oxygen and establish a free airway.

3. For convulsions give a short-acting barbiturate intravenously, e.g. pentothal.

4. For a severe fall in blood pressure, give Methedrine.

The toxic effects appear formidable, but I do not feel that they should preclude this valuable analgesic therapy which is, in my opinion, quite safe if a preliminary barbiturate is given one hour before, and the case is 'specialled' from a nursing point of view.

Perhaps one of the most heart-rending sights in hospitals is that of a child crying because of pain, a situation which is seldom, if ever, necessary. If the clinician is disinclined to use intravenous Procaine therapy for burns in childhood, there is another treatment which I have found very useful for initial cleaning and changing dressings. This is the rectal administration of Surital (P. D. & Co.).

The details are as follows:

1. A 5% solution of Surital is prepared using normal saline or distilled water.

2. Dosage is based on the weight and physical condition of the patient and varies from 0.8 to 1 g. per 50 lb. body weight. Routinely, the writer gives 0.25 c.c. of this solution per lb. body weight.

3. Induction generally ensues within 5-15 minutes of administration.

4. Respiratory tree spasm is rare but it is wise to have a B.L.B. mask and oxygen near at hand.

4. SYSTEMIC ANTIBIOSIS

In an attempt to prevent, or at least modify, infection, general antibiosis is indicated. I would suggest a rapidly released crystalline penicillin 500,000 units as an initial dose followed by a slow-release penicillin 400,000 *b.i.d.* and streptomycin 0.5 g. *b.i.d.* for 7 days.

At the end of this period, this antibiosis should be reviewed. If the infection is controlled, as judged by the temperature and pulse charts and the appearance of the burnt areas, it should be stopped and only renewed if a surgical procedure is contemplated which will disturb the protective granulation tissue of the lesion.

If the general and local signs suggest continued infection, then swabs should be taken from representative parts of the lesion and the response of the infecting bacteria to penicillin and other antibiotics determined to guide future antibiosis.

5. INITIAL LOCAL TREATMENT

Although one has to accept the usual contention that the burning agent sterilizes the burnt

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area, there is no doubt that the presence of tissue debris lends itself to early infection. Therefore all burns should be surgically cleaned with removal of skin debris, blisters, clothing, etc. as soon as possible. Saline swabs are often advised but the writer prefers 1% Cetavlon soaked swabs; the Procaine intravenous therapy supplies an amenable patient for this cleaning procedure.

At present there are two methods of treatment in popular vogue—the so-called open or exposure method and the treatment where the burnt area is closed from its external environment by dressings. Most clinicians feel that it is wrong to be a bigoted subscriber to either method and that the peculiarities of the burnt area, and of the patient as a whole, dictate which method should be used. The clinician must be prepared at any stage of treatment to revert to the other method if circumstances so dictate. Experience seems to indicate that the exposure method is the more successful as a treatment of superficial burns and the closed method is indicated in the following circumstances:

1. Encircling burns, especially those where a tourniquet effect may ensue. The greasy component of the dressing seems to soften the resultant hard eschar.
2. Full thickness burns.
3. Burns of the ear and other sites where there is minimal subcutaneous tissue.
4. Burns of digits.
5. Burns of areas which must bear the effects of the body weight of the recumbent patient.

Generally speaking, then, the depth of the burn plays a big part in the choice of treatment and therefore the clinician must be able to make a fairly accurate assessment of the patient's burns from the point of view of depth of skin damage. The following clinical observations will assist in this important assessment:

After removal of debris, etc. the superficially burnt area reveals evidence of hair follicles, sweat gland orifices and accumulations of skin pigment.

Testing of the burnt area with a sterile hypodermic needle reveals pin prick sensitivity in superficial burns.

The area is moist and has a shiny, granular appearance in marked contrast to the dull surface of a deep burn.

When the deep burn is cleaned, a white, coagulated, anaemic area is revealed, dry and dull and insensitive to pin prick stimulation. Unfortunately, several burns (and even areas in the same burn) cannot be accurately assessed by these criteria; but this does not vitiate the general conception. Treat these burns (other factors being equal), by an exposure method and re-assess after 7-10 days. By this time a very accurate assessment is possible. (See subsequent notes on *Crusting*).

In small, circumscribed, full thickness burns (e.g. those caused by molten metal splashes, electrical burns, etc.) which initially reveal their depth, primary excision of the dead tissue and immediate grafting is indicated; but a note of caution must be sounded regarding this treatment, in order to prevent removal of useful tissue. The clinician must not excise tissue unless he is sure that the tissue is totally dead.

(*a*) DETAILS OF THE EXPOSURE METHOD

1. Clean the area completely with 1% Cetavlon.
2. Dry the area gently with sterile towels.
3. Frost the area with penicillin in lactose powder and repeat 4-hourly until the area is completely dry.
4. Protect the exposed area from bed clothes with clean, freshly ironed sheets over the large bed cradles. These cradles should be lashed fast to the bed-side so that their accidental collapse is prevented.

(*b*) DETAILS OF THE CLOSED METHOD

1. Clean the burnt area completely with 1% Cetavlon.
2. Frost with penicillin in lactose powder.
3. Cover completely with 'greasy' dressings.
4. Cover with adequate cotton wool and ensure an even pressure by carefully applied pressure bandages.
5. Elevate and splint injured areas as possible and necessary.
6. Change dressings not more frequently than at weekly intervals. Indications for changing of dressings are:
   (*a*) Distal oedema due to excessive local pressure.
   (*b*) Seepage of serum, etc. through the dressing.
   (*c*) Slipping of the dressing.
   (*d*) Unexplained occurrence of increased pain or generalized toxic effects.

Greasy Dressings. These have the advantages of ease of application, ease of removal without damage to young granulation tissue, and are supplied commercially in sterile tins. Tulle gras is the form which is usually favoured but, in my opinion, the excess of paraffinum molle tends to lead to tissue maceration and impedes skin regeneration. (Excessive grease of the paraffin type is abnormal as a skin application).

I feel that adequate trials of the following preparation should be made in place of tulle gras—viz. tulle or wide meshed sterile gauze coated with Glasgow Burn Cream, viz.

Cetyl Trimethyl Ammonium Bromide	1%.
Ol. Ricini	25%.
Bees wax	1.8%.
Wool fat	1.8%.
Cetyl Alcohol	5%.
Glycerine	10%.
Water	52.4%.

(*c*) A NEW TECHNIQUE USING NOBECUTANE (BOFORS OF SWEDEN), WHICH EMBODIES THE ADVANTAGES OF THE EXPOSURE AND CLOSED TREATMENTS

The writer has experimented with several cases of burns and scalds following the general

principle that these lesions heal most readily if the area is kept dry.

The following points are relevant:

A. *Superficial Burns.* As soon as the general therapy against shock is under way the burnt areas are:

1. Cleaned with 1% Cetavlon swabbing under anaesthesia and in the operating theatre.
2. Examined to confirm the superficial nature of the burn.
3. Lightly powdered with penicillin in lactose powder.

Theoretically, this ensures a thin film of the antibiotic under the thin film of the Nobecutane; also recent experimental evidence has suggested that a high local concentration of a hexose is beneficial for epithelialization.

4. The whole area is sprayed with Nobecutane solution with a generous overlap onto contiguous normal skin, and the film is allowed to dry.
5. The area is protected by clean sheets and large bed cradles.

It is interesting that the rapidly evaporating solvent of Nobecutane seems to coagulate the exuding serum very quickly, and the total exudation is minimal in comparison with that occurring with other methods of treatment. The 'burn cover' is made up of the Nobecutane film superjacent, and intimately adherent, to a coagulated serum layer; the whole incorporating a fine contact of penicillin powder. In essence, the treatment is a drying technique, using an artificial covering over a surgically clean wound and also embodying the advantages of an exposure technique.

Pain is greatly relieved and the results suggest that the method is a boon and a blessing in paediatric burns and scalds.

The child thrives, takes its feeds, sleeps well and does not have to face the frightening ordeal of dressing changes, etc. The burn 'cover' wrinkles and expands with essential body movements, e.g. respiration, and, in normal circumstances, the film remains intact.

In none of our superficially burnt cases has any pus or fluid collected under the surface covering, nor has there been any edge sepsis. The method was given a severe trial in the case of a child with burns of the buttocks—the only other factor of note in its treatment was 'gallows' suspension. This child healed rapidly and without sepsis.

Surface epithelialization proceeds apace under the film (our impressions are that the healing in superficial burns so treated is much faster than that in cases treated by the classical exposure method) and the dry surface cover peels off easily.

B. *Deep Burns.* A small pilot series of deep burns has been treated in the manner described above for superficial burns. The results were interesting. There was initial success in that the burnt area was quickly dry, no pus formed, pain was minimal, there was less serum loss and the patient was very comfortable. But the covering, which consisted of a Nobecutane film, coagulated exudate and dead tissue resembled the dry plaque which formed in the days when tannic acid treatment was in vogue. The formation of a line of demarcation between the plaque and the living tissue appeared between 12 to 14 days, and its approach was manifested by accumulations of clear serum under the plaque and between it and the coagulated serum and Nobecutane layer.

When the dry plaque was peeled off by a modified sloughectomy procedure, a clean, non-infected area was exposed—ready to take skin grafts.

It has been possible in certain cases to get some patients ambulatory during treatment, wearing sterile gowns or shirts, with consequent uplift in their general health and morale.

A sufficient number of deep burns has not yet been treated to draw firm conclusions, but my personal impression is that the area left after this Nobecutane treatment is dry, cleaner and more suitable for grafts than the moist, actually or potentially infected area left after the conventional 'closed' technique.

LOCAL TREATMENT OF BURNS IN SPECIAL SITES

The Face. All burns of the face should be carefully cleaned and treated by the exposure method. A coagulated crust forms and in superficial burns this surface layer soon begins to separate. During this process the stiffening of the normally mobile facial tissues is uncomfortable for the patient and can be relieved by frequent thick applications of Nivea cream. Crusting of the nostrils and lips requires gentle removal with hydrogen peroxide and applications of liquid paraffin.

Crusting and stiffening of the eyelids prevent these structures from protecting the eyeballs. In superficial burns the crusts should be removed constantly and smeared liberally with Nivea cream or liquid paraffin. In deep burns a plastic surgical opinion should be sought early regarding the countering of scarring effects, e.g. ectropion; 5% Albucid drops should be instilled into the conjunctival sacs every 4 hours to counter local sepsis. The presence of corneal lesions requires 1% atropine eye drops daily, to counter iris adhesions and blockage of the filtration angle.

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Ears. Burns of the external ear do very badly, with sloughing and scarring—primarily due to an ischaemic pressure effect of accumulated 'inflammatory products', e.g. oedema in the virtually non-existent space between skin and cartilage. The following régime is suggested for their treatment—clean, powder, greasy dressing, plenty of wool and a pressure bandage against the skull to counter excessive local swelling.

Mouth and Pharynx. Burns of this area may be complicated by potential or actual oedema of the glottis, and *early* tracheotomy may be indicated.

Flexure Areas, e.g. Neck, Axillae or Groins. Burns in these areas, especially the neck, heal badly. The chief aid to countering this is immobilization of the area concerned. This may require a degree of ingenuity in any particular case; head traction, a modified Minerva plaster of Paris, neck extension over subscapular pillows, etc. are helpful methods. Neck burns should be grafted with broad sheets of thick split skin grafts fixed in position by fine sutures.

Extension and splintage are useful fixation methods for groins and axillae.

Pudendal and Perineal Areas. The male and female genitalia tend to swell easily after irritation from any cause, and burns are no exception. The areas should be shaved during the preliminary cleaning and treated by an exposure method. A catheter should be inserted and the dependent scrotum elevated. Perineal and pudendal burns are best treated by the Nobecutane technique and the areas washed with 1% Cetavlon after defaecation.

Small children, and even adults, are easier to nurse on the gallows suspension.

Burns of Digits. The anatomical peculiarities of the digits are such that early immobilization of the tendons in the sheaths and of the small joints (especially the metacarpo-phalangeal) is prone to cause stiffness after burning injuries. This is due to oedema, rigidity of, and possibly strangulation by, the coagulum, and absence of proper functional rest of the joint.

Extensive and deep burns should be treated by the closed method, each digit should be bandaged separately and the whole hand supported in the position of physiological rest on a plaster slab. The whole limb should be elevated and thus maintained for at least one week.

Active, purposeful movements should be encouraged *in situ.*

The first dressings should be done on the seventh day. Superficial burns should be cleaned and redressed and increasing movements encouraged. Deep burns over tendons and joints should not be subjected to sloughectomy lest these important structures are inadvertently damaged.

The sloughs should be separated by the aid of proteolytic preparations or Eusol and the areas grafted with full thickness grafts.

6. PROBLEMS OF INTERMEDIATE CARE

(*a*) *Nutrition.* Diet should be of a good mixed nature with special emphasis on a high, first class protein content.

Adjuvant doses of vitamin preparations are essential—especially vitamins A, B complex and C.

Regular bowel actions may be difficult because of postural problems and care, in the form of adequate dietary roughage, laxatives or enemata, should be given full attention.

Adequate fluids by mouth are essential.

(*b*) *Blood Estimations.* These should be made at least twice weekly in order to detect and counter anaemia.

The writer feels that it is essential to maintain the blood volume, haemoglobin, red cell count, etc. up to the normal readings for the particular patient by fresh blood transfusions.

Low normal and 'near normal' readings cannot be accepted. Whipple and Robbins[1] state:

> 'The prevention of anaemia in major injuries undoubtedly facilitates the prevention of hypoproteinaemia'.

Clarke[2] writes:

> 'May not the red blood cells contain as much as, or more than, the constituents of the plasma, of the raw materials—the bricks and mortar of tissue repair? Perhaps the development of post-traumatic anaemia, even after adequate blood transfusion, is partly due to removal of red cells for this purpose of repair.'

There is much to be said for this conception of the dynamic role of red cells in injury. These cells are not just inert haemoglobin carrying entities and their analysis has shown that they contain a large variety of biochemical substances (not concerned with oxygen and carbon dioxide carriage) including nucleoproteins, peptides, catalysts such as phosphatases, carbonic anhydrase, co-enzymes, lipase, urease and choline esterases. The advancing capillary buds of granulation tissue carry a red cell content which may have this constructive function; perhaps it also explains the function of diaphoresis.

Whether this form of theorizing is correct

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or not, there is no doubt that patients in whom the post-traumatic anaemia is successfully countered do very much better, heal quicker and have a bigger percentage of successful skin grafting. Conversely, the reverse type of patient is usually the one who, after holding his own for some time—even months, suddenly goes downhill and dies rapidly—and the autopsy findings do not explain the cause of the rapid deterioration.

In nuclear warfare burns, correction of this post-traumatic anaemia will be a major factor in the intermediate phase of treatment.

(*c*) *Problems of Crusting over the Burnt Areas.* Careful observation of the type of crusting in the burnt area will often confirm, or otherwise, the original estimation of the depth of the burn.

It appears that when superficial burns are treated by the 'exposure' method, a thin crust forms. This is dry, any subjacent fluid is serous, painless to palpation, and *no* pus is present. It separates (beginning at the edges) and there is a healed dry skin underneath.

At present it seems wise to leave this crust completely alone. It is futile to start applying Eusol-soaked dressings to this natural protective covering. Eusol is useful only for expediting sequestration of dead (and usually infected) tissue. The crust is not a tissue but a dried exudate.

In cases where deep burns have been treated by the exposure method, it seems to the writer that crusts which form are very thick, irregular and slowly increasing in thickness, wet and often pus soaked (the pus need not be entirely due to infection, but is also due to the ordinary sequestration mechanism of dead tissue separation). It is the writer's impression that these crusts with sloughs usually fragment into smaller areas.

These types of cases are candidates for Eusol soaks, which are designed to speed up natural sequestration and reveal fresh young granulation tissue areas ready for skin grafting. It is necessary to point out that prolonged applications of Eusol to young granulation tissue tend to age the capillary-fibroblast elements and thus render this tissue less favourable for successful skin grafting.

Burns treated by the closed method usually begin their sequestration process more slowly and the Eusol therapy is indicated as soon as it is apparent how much tissue is going to separate.

Intermediate depths of tissue damage may reveal another feature—nodularity of the crust. This appears about 7 days after injury and seems to indicate that natural and spontaneous epithelialization will occur. If the crust is peeled off, the late forming nodules are found to have been superimposed over actively growing islets of surviving epithelial skin elements.

The Problems of Contracture Formation. In the case of limb and flexure burns these are very important and present many difficulties. The factors involved are not yet completely understood, but the following points are relevant.

1. Prevention of contractures by immediate treatment is such easier than their cure.
2. The wound which contracts most is the open one with full thickness skin loss which is left to heal in from the periphery.
3. Burns, where the corium layer is totally destroyed and the raw surface is covered with very thin split skin grafts, have a marked tendency towards contracture. The writer feels that the corium layer must be replaced as adequately as the circumstances permit, by full thickness grafts ideally, or at least by thick split skin grafts.
4. The linear fibrous areas which result after spaced island grafting have a tendency to late contracture.
5. The limbs and the cervical region must be splinted in the physiological position of rest right from the commencement of treatment.
6. Early physiotherapy with occupational therapy is an essential element in the management of burns.
7. The junction of the graft and the margin of the recipient area must be placed where the resultant scars are exposed to the least possible tension, i.e. in normal crease folds and at the lateral margins of joint surfaces.

7. SKIN GRAFTING

In most cases thick split skin grafts (i.e. containing a layer of corium) are the most useful, and obviously the most plentiful if a big recipient area has to be covered.

The burnt areas should be grafted within 3 weeks of the initial injury. Sloughs which become demarcated and have begun to separate should be assisted to do so by sloughectomy procedures. The writer has not been impressed by the results of proteolytic agents for assistance in this slough separation, but the Trypure Novo preparation (Evans Medical Supplies Ltd.) promises more efficacy than the others. Immediate skin grafting after sloughectomy seems unwise in the light of present experience; 3 or 4 days of cleaning up procedures with Eusol dressings before grafting seem to yield the best results. These remarks do not apply to the raw areas left after removal of the Nobecutane sloughs.

It is difficult to be dogmatic about the following point, but the writer feels that the

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skin grafts should be placed in edge apposition and not spaced by corridors of granulation tissue. These latter never seem to be covered adequately by skin edge outgrowth, they seem to be stimulated to excessive granulation after wound grafting, are very difficult to graft subsequently and, when they do at last heal, they form thick strips of scar tissue. Apart from these criticisms, the patient regards these raw areas in the light of 'the wound has still to heal'.

Anchoring of the grafts is a *sine qua non* and may be obtained in several ways.

(*a*) It is occasionally possible by direct marginal suture.

(*b*) In awkwardly shaped sites Stent moulds may be used to support grafts.

(*c*) Most often adhesive tulle gras dressing, reinforced by cottonwool padding, pressure bandages and/or plaster of Paris, is used.

In this connexion, it is the writer's impression that the tulle gras should retain only the minimum of vaseline grease—because excessive grease seems to promote skin surface maceration in the grafts and the circumrecipient area. He feels that tulle gras violates the principle that dryness is essential for skin healing, and matters become much worse if a degree of local infection is also present.

All split skin grafts seem to survive in the serous exudate of the recipient area for the first 48 hours. Then, if the recipient area is infected to any degree, the wound becomes very moist and some proteolytic destruction of the grafts occurs with much local fluid accumulation. This process seems to be raging at its peak about the fourth day. A possible result is complete destruction of all grafts and a proliferation of Proteus, Pyocyaneus and Streptococcus organisms in the wound. Experience would suggest that all this is more pronounced when tulle gras is the dressing. These points are of some practical interest with particular reference to timing of post-operative dressing of the wound. It is very difficult to identify latent infection and thus sub-divide recipient areas into categories of clean and relatively dirty wounds. All grafts are in their most vulnerable state between the second and fifth days after operation, and yet this is the period with infected grafts in which something must be done to preserve living remnants.

To overcome this problem the writer has tried, in suitable cases, to frost with penicillin in lactose powder and expose the grafts to the atmosphere by enclosing the grafted area under cardboard boxes held in place by Elastoplast. Partial success has attended these methods but crusting around the graft edges has been a problem.

Glueing of the grafts by Nobecutane and exposure has been successful in selected cases, and the method has been put to the test of really suspect recipient areas. The writer is very impressed with skin graft fixation by this method and uses it exclusively for grafting of flat surfaces.

Another régime that was tried was dressing the wounds after 48 hours, removing the tulle gras, frosting with penicillin in lactose powder, dressing with gauze wrung out in 1% Cetavlon or Merthiolate, cotton wool padding, pressure bandages and splintage, and leaving the whole alone for 14 days. This has been attended by considerable success provided the dressing was done by the surgeon himself.

(*d*) The ideal for skin grafting dressings, which can be left undisturbed for 14 days or more, i.e. until sound healing of the grafts, embodies the following virtues:

1. Protection from external forces which may move the grafts on the recipient area.
2. Ability to change these dressings without disturbing the grafts.
3. Some absorptive mechanism which will keep the grafted area dry.
4. The dressing surface should be non-water-wettable and non-toxic, and the whole dressing should be capable of easy sterilization.

A trial supply of Curity Telfa Non-Adherent Dressing Strips (manufactured by Bauer and Black, Chicago) was obtained from their South African agents. Great success attended this trial and these dressings satisfy the above criteria. The only criticism is that the dressing strips supplied are too small and much larger ones are required for grafting procedures. Their routine usage appears to be economical.

8. A SCHEME FOR THE TREATMENT OF BURNS

A. INITIAL STAGE (FIRST 3 DAYS)

1. Admit as *urgent direct admission.*
2. Relieve pain:

(*a*) In slight cases, by morphine gr. ¼ plus Daptazole mg. 25 intramuscularly, for the adult.

(*b*) In severe cases, by Procaine intravenous drip in one arm or leg, after a preliminary Barbiturate.

3. Start plasma intravenous drip in the other arm or leg and take a sample of blood for compatibility tests. Assess shock clinically and consider need for noradrenaline and hydrocortisone.
4. Check the respiratory passages and look at the mucosae for cyanosis.
5. Undress the patient completely and estimate the extent of the burnt area. Examine for other

injuries and assess their importance in the overall clinical picture. Wrap the patient in a sterile sheet.

6. Estimate the quantity and types of fluids to be given and adjust intravenous drips accordingly. In cases where there is extreme shock, consider whether an intra-arterial blood drip may not be the best plan.

7. Insert a catheter, arrange for hourly collection and measurement of urine; institute fluid balance charts, half hourly temperature, pulse and respiration charts and begin antibiosis.

Tetanus in burns is not rare, so it may be wise to give a prophylactic dose of anti-tetanic serum.

8. At half-hourly intervals re-assess the clinical condition and fitness for theatre procedures.

9. Take the case to the theatre, clean the burns with 1% Cetavlon, assess the depth of the burn and confirm the extent. Record on clinical notes.

Decide on exposure or closed treatment and perform it. Decide on the mode of splintage, where necessary. Return to the ward.

10. Check the patient's clinical state and especially urinary output and the specific gravity of the urine. Adjust intravenous therapy in the light of these findings.

11. Try oral fluid feeding and, if refused, give it by a soft nasal tube.

12. The patient should be 'specialled' by doctor and nurse during the first 72 hours of treatment with particular reference to shock, fluid balance, urine output per hour, quantity of oral feeds, including light fluid nutritious diet with a high protein content.

13. When shock is combated and urinary output is satisfactory, remove the catheter.

B. INTERMEDIATE STAGE (4TH-14TH DAY)

1. Check the burnt area—look for cracking of coagulum, fluid seepage, smell, distal oedema, watch for thickening and 'pseudo barnacle formation' of the coagulum in those burns treated by the exposure method. In burns treated by the closed method, remove dressings under anaesthesia between the seventh and tenth days and re-assess where a demarcation line has formed. Estimate when a sloughectomy procedure can be countenanced.

2. Every fourth day, estimate the red cell count, haemoglobin, blood proteins and electrolytes and correct by intravenous methods any anaemia or deficient reading.

3. Check the diet, fluid intake, daily urine output, frequency and actual nursing details of defaecation.

4. After 7 days of antibiosis, check the necessity for this therapy and, if it should be prolonged, select an antibiotic by sensitivity tests.

5. During this intermediate stage, assess whether the patient can get out of bed and arrange for physiotherapy.

C. FINAL STAGE (15TH-21ST DAY)

1. Continue all observations annotated under *Intermediate Stage.*

2. The superficial burns should be healed completely during this stage.

3. The deep burns should be well demarcated and the removal of dead tissue should be accelerated by sloughectomy.

4. By the very latest, these areas should be ready for skin grafting procedures by the end of this period.

When the burnt areas are healed, intensive physiotherapy and specific occupational therapy should be ordered to complete the treatment.

CONCLUSION

As stated in the preamble, this paper records the findings of a sustained, clinical interest in patients suffering from burns, gathered from a long and unique experience of this type of injury.

The writer believes that several of the points suggested in the treatment are original.

He has avidly read the voluminous literature on this subject which has accumulated over the years, and thus it is well nigh impossible accurately to quote references to papers which may have influenced his attitude to the many facets of this very interesting problem. However, in the text, proper appreciation has been paid to Messrs. Allen & Hanbury's *Pyrogram Ready Reckoner for Fluid Therapy and Urine Output,* and to the manufacturers' instructions concerning noradrenaline (Levophed) and Methedrine administration.

OPSOMMING

Hierdie referaat bestaan uit 'n opsomming van die skrywer se 17 jaar van ondervinding van, en sy intense belangstelling in die probleem van die behandeling van brandwonde wat deur droë en vogtige hitte veroorsaak word.

Dit het hom tot die gevolgtrekking laat geraak dat, vir doeltreffende beheer, daar een belangrike vereiste is, nl. 'n behoorlik gereguleerde skema van optrede wat die hele behandeling van die slagoffer se beserings insluit, en wat geleer en toegepas kan word deur die verpleeg- en die junior mediese personeel tot voordeel van die pasiënt, en met ekonomiese gebruik van hospitaalakkommodasie. So 'n skema word dan ook in hierdie referaat uiteengesit.

Die behandeling van brandwonde word vandag beïnvloed deur die heersende mediese modes en die grille van diegene aan wie se sorg die pasiënt toevertrou is, daar is geen eenvormigheid nie, en gevolglik is dit moeilik om die werklike en betreklike resultate na waarde te skat.

Die bestuur van die pasiënt word in drie afdelings verdeel, nl. die onmiddellike, die intermediêre en die finale of herstel-behandeling.

(*a*) *Ommiddellike Behandeling.* Die hele probleem van vloeistofterapie word breedvoerig bespreek, en presiese inligting oor die soort en die hoeveelheid vloeistowwe wat nodig is, word vir oorweging voorgelê. Die noodsaaklikheid van kortstondige maar doeltreffende binne-aarse terapie word benadruk. Die verligting van skok en pyn word breedvoerig bespreek.

In die verbygaan noem die skrywer die huidige konvensionele blootstellings- en geslote metodes vir aanvanklike plaaslike behandeling, en verstrek dan besonderhede oor 'n gewysigde alternatiewe metode

wat die voordele van albei bogenoemdes meebring. Kortom kom dit neer op die bedekking van die beskadigde oppervlaktes met Nobecutane (Bofors, van Swede). Hierdie terapie bestry voortdurende deursypeling en stel die pasiënt in staat om reeds op 'n vroeë stadium uit die bed uit op te staan en rond te loop, met al die voordele wat dit vir sy algemene welsyn en moreel meebring.

(*b*) *Intermediêre Behandeling.* Die probleme voortspruitende uit skynbaar onvermydelike en kwaadaardige bloedarmoede en ondervoeding word breedvoerig bespreek. Plaaslike behandeling bestaan uit die verwydering van sekwestrasie-weefsels, en voorbereidings vir die vervanging van die vel. Metodes vir die vasstelling van die diepte van die brandwond word aan die hand gedoen.

(*c*) *Finale Behandeling.* As 'n pasiënt op die regte manier behandel word, behoort die beskadigde oppervlaktes teen die einde van die derde week, op sy laaste, gesond of gereed vir huidinplanting te wees. Die toepaslike probleme wat deur die huidinplantings-proses opgelewer word, word bespreek, en 'n kritiese oorsig van bevredigende verbindsels vir sodanige inplantings word verstrek.

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BURNS IN CHILDHOOD

ANALYSIS OF THE CAUSES AND MORTALITY IN 578 BURNS ADMITTED TO A GENERAL HOSPITAL IN 3 YEARS (1953-55)

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The problems of thermal injuries are a constant challenge to surgical treatment, and the mental and physical suffering they bring in their wake requires highly skilled and sympathetic nursing. They constitute, too, a factor of considerable economic importance in terms of hospitalization and the social care and rehabilitation required for the disabilities of the survivors.

Colebrook, whose authoritative views have done so much to focus attention on the causes and prevention of burns and scalds, has estimated that 10,000 children with these injuries are admitted to British hospitals annually and 50,000 adults are treated as in-patients and out-patients in equal proportion. The loss to industry and schooling is of the order of 1,000,000 man-days, and approximately 200,000 schooldays per year.[4, 7] In this country, where the dangers to which children are exposed are more in evidence, the figures must be proportionately higher. At this hospital alone nearly 200 children under 10 years of age are admitted annually with burns; and the frequency of old infected burns and late contractures admitted gives some indication of the number that do not come to hospital.

The biggest single cause of child mortality in Britain and the U.S.A. is trauma,[1] and the same is probably true of White children in this country, where living conditions are of a similar pattern. In the U.S.A. burns are second in importance to road accidents as the major cause of deaths in children under 15 and in 1948 30% of the 8,000 annual burns deaths occurred in children.[1] In England over 40% of all burns deaths occurred in children.[4, 7] Probably a comparable figure obtains in this country in White children where infectious diseases have almost been eliminated as major causes of child mortality. In the U.S.A. each year twice as many children die from burns as there are actual cases of poliomyelitis.[12]

This is far from true, however, among non-White children where gastro-enteritis and tuberculosis still take a heavy toll. Nevertheless, trauma ranks high after these as a cause of death.

Furthermore, of all deaths in South Africa due to road accidents and burns in the years 1952-54 inclusive, 10% of White deaths were due to burns whilst 32%, 34% and 33% respectively were the figures for African, Indian and Coloured deaths from burns.*

* Bureau of Census and Statistics, Pretoria 1956, personal communication. The figures for the non-Europeans refer to urban areas only.

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CAUSES OF BURNS AND SCALDS (TABLE 2)

Since burns and scalds represent such a common form of trauma, it is worth examining some of the causative factors which contribute to this state of affairs.

An analysis has been made of the causes of burns and scalds in children admitted to a large general hospital during the last 3 years. In the large majority of cases the notes on admission have indicated the type of accident. Despite the brevity of some charts and the fact that nearly 20% of these injuries were not ascribed to a specific agent but simply marked 'burnt' or 'scalded', the remainder give a reasonably accurate picture of the role of different factors in the 3 age groups chosen, 0 to 1, 1+ to 5 and 5+ to 10 years. These groupings have been selected rather than 0 to 1, 2 to 5 etc., as many case reports have inaccurate records of age due to the unavoidable ignorance of the parents. Fewer inaccuracies will result by using 0 to 1 as the first group and to include all children over 1 year old and under 5, in a 1+ to 5 years group.

The majority are due to unwitting carelessness on the part of the adult family who care for the child, and their ignorance of the dangers of modern appliances. Among non-Whites the root cause is undoubtedly the poverty-stricken home where the combination of large families crowded into small ill-lit rooms dependent on open fires or Primus stoves for cooking, and using large unguarded pots of food, make these accidents inevitable. Most of the African and Indian patients served by this hospital live in such slums and shanty-towns in and around the city.

Fifty-five per cent of African and 40% of Indian burn admissions to King Edward VIII Hospital are children under 10 years of age (Table 1).

TABLE 1: INCIDENCE OF BURNS IN CHILDREN UNDER 10 YEARS ADMITTED TO KING EDWARD VIII HOSPITAL (1953—1955)

	Age (Years) 0 *to* 1	1+ *to* 5	5+ *to* 10	*Total Admissions under 10 Years*	*Total All Burn Admissions*	% *under 10 Years*
African	100	274	130	504	914	55%
Indian	8	39	29	74	182	40%

The high percentage of scalds is shown in Table 2; 74% of burns in African children were due to boling water (the commonest cause), tea and coffee, whilst the number of porridge burns conjures a dramatic picture of the difficult home environment of these children (Table 2). At the Birmingham Accident Hospital, of 259 patients of all ages admitted, 71% were children under 10 years of age, and of those 61% were due to scalds.[4] In one series of 84 burns in the U.S.A. 15% were due to scalds,[1] and in a second series of 344 cases only 8% were due to scalds.[2]

Nearly 75% of all scalds of both African and Indian patients admitted to this hospital occurred in children under 10 years, but it is interesting to note a significantly smaller proportion of scalds among Indian children. This difference is represented by the greater number of burns in the Indian children caused by accidents around fires and is twice that of the African admissions (38% Indian children, 19% African children; Table 2). No doubt the near-naked African child is exposed to the chance of scalding while the more fully clothed Indian children are in greater danger near fires and the open flames of lamps and heaters; particularly the girls whose long saris make them potential funeral pyres in these circumstances.

Burns due to Primus and paraffin stoves, and oil lamps, form an important group of causes among children (20% African, 14% Indian) and many of the most severe burns are due to these highly inflammable heating and lighting agents.

While no children were burnt in the 3 years under review by agents listed in the miscellaneous group, it is not uncommon among teenagers and adults to find burning as a means of assault in Africans and suicide in Indians.

EXTENT OF BURNS

Obvious difficulties present themselves in analysing burn areas from case notes.

The extent of surface area burn can only be accurate if particular care has been taken to estimate and chart the injured area; if areas were described and not charted diagrammatically then at best only a rough assessment could be made. In the analysis of these burns many cases were described briefly and sometimes loosely. Where the description was too brief or couched in vague terms the maximum assessment for the area was computed and the case grouped accordingly, e.g. 'burn of arm' (with or without diagram) was assessed at the full extent of the arm, 8%. It was hoped that by this method and in conjunction with the

TABLE 2: CAUSES OF BURNS IN CHILDREN ADMITTED 1953—1955

		Age in Years 0 to 1	1+ to 5	5+ to 10	Admissions under 10 years	Total All Admissions	% All Admissions under 10 Yeras	% Frequency under 10 Years	% Frequency All Ages
1. *Scalds* Water Tea Coffee Soup Curry *Porridge	African ..	54	165	86	305	425	72%	74%	58%
	Indian ..	6	19	10	35	48	73%	56%	29%
2. *Burns* (a) *Fire:* Fall Wood Coal Clothes	African ..	17	41	23	81	158	50%	19%	22%
	Indian ..	3	6	15	24	69	38%	19%	41%
(b) *Inflammable:* Oil Primus Benzine Petrol Paraffin	African ..	8	10	5	23	109	20%	5%	15%
	Indian ..	0	3	1	4	29	14%	6%	17%
(c) *Miscellaneous* Chemicals Lightning Assault etc.	African ..	—	—	—	—	19	—	—	3%
	Indian ..	—	—	—	—	20	—	—	2%
3. *Unknown*	African ..	21	57	15	93	186	50%	18%	20%
	Indian ..	—	6	3	9	16	56%	13%	9%
*Porridge	African ..	4	21	6	31	48	63%	8%	7%

other notes of the case the error would not be in the direction of under-assessment. In all cases of death an accurate assessment was obtainable as the Government Pathologist's reports contained accurate written and pictorial descriptions.

Ten per cent surface area burn is taken as the extent below which many cases can be treated as out-patients and above which there is some degree of surgical shock and hospital treatment is necessary.

Eighty-four per cent of the African children admitted had burns of under 10% and the comparable figure in Indian children was 59%. In the series quoted by Bull and Fisher 55% of all burns seen were under 5% and a further 34% between 5 and 15%.[3]

This high figure indicates that the majority of burns, about 4 out of 5 cases, are small in extent and adequately trained nursing and medical personnel can deal with many of these as out-patients, even with poor children. The African and Indian mother will go to great lengths to follow carefully explained instructions, provided that they understand their purpose. The use of a closed dressing with plaster of Paris reinforcement for limbs enables patients to go home and return for daily injections of penicillin and inspection of the burn in 10 days. Re-assessment can then be made for any full thickness loss and therefore the need for grafting.

FREQUENCY OF BURNS IN DIFFERENT AGE GROUPS

Fig. 1 indicates that the 1-5 years age group is the peak period for the occurrence of these injuries. This is to be expected as children at this age are beginning to walk and stray from parental control. The fall-off amongst Indian children is, however, not so great in the 5-10 years age group. In a series of 259 cases at Birmingham Accident Hospital 80% of the injuries occurred in the 0-4 years age group and 20% in the 5-10 year group.[4]

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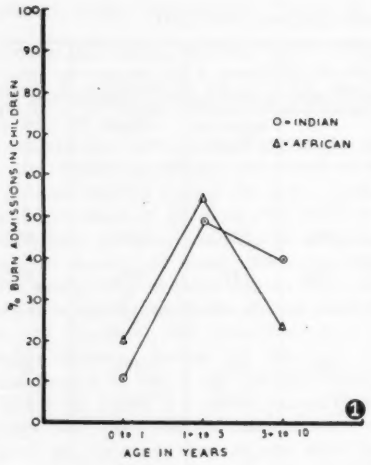

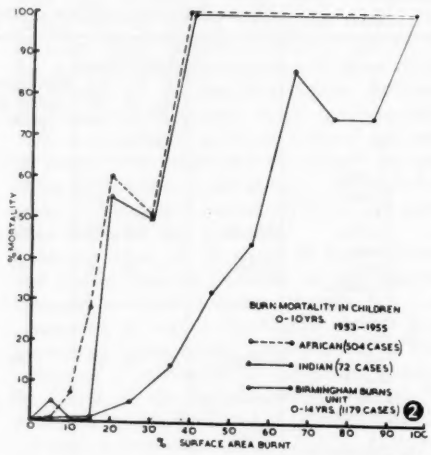

DISTRIBUTION OF BURNS

Table 3 indicates the distribution of burns and scalds in children in 3 areas of the body divided arbitrarily into upper (including head and arms), trunk, and lower (including buttocks). Nearly half of all the burns involved the lower limbs, buttocks and perineum. There was a progressive increase in the number of burns involving lower limbs and buttocks as the child grew older (35% to 60%) with a corresponding decrease in both trunk and upper limb burns.

TABLE 3: DISTRIBUTION OF BURNS IN CHILDREN 0-10 YEARS

Years	*Face, Head, Neck, Arms*	*Trunk*	*Buttocks, Perineum and Lower Limbs*	*Scattered*
0 to 1	33%	13%	35%	20%
1+ to 5	29%	11%	47%	13%
5+ to 10	23%	8%	60%	9%

Overall Distribution in Children 0—10 Years:
48%—lower limbs, buttocks and perineum.
30%—upper limbs, face and neck.
12%—scattered burns.
10%—trunk burns.

MORTALITY

Fig. 2 indicates the high mortality that burns exact in children treated in a busy general hospital where no special facilities exist. The figures are contrasted with those of a highly organized centre for burns treatment at Birmingham Accident Hospital and indicate what proper care and skilled attention can achieve.

In the 578 children in this series the over-all death rate for those with 20% area burns (little more than 1 lower limb in extent) was over 50% in both African and Indian groups. The comparable figure from the Birmingham Accident Hospital is less than 5%. Burns of over 30% surface area left no survivors in this hospital, whilst at Birmingham the death rate for this degree of burn was 12-14%[3] (Table 4).

There was a significantly higher mortality in the 0-1 African age group (Table 4) where a 50% mortality in burns over 10% body area and a 100% death rate in those with 15-20% burn area occurred. The Birmingham figures group the 0-4 years together, but Bull and Fisher state that there is no appreciable difference between younger children (0-4 years) and older ones.[3] The figures for Indian children were too few for a significant analysis.

Not only is the mortality higher in the 0-1 years age group here, but it appears that the 1-5 age group of African children has a higher mortality than the 5-10 age group. In this latter age group there were no survivors with burns above 30% area. For those with a burn between 15% and 30% surface area there were no deaths in 5 patients, whilst in the 1-5 age group there were 9 deaths in 14 patients for this size burn (Table 4).

FACTORS CONTRIBUTING TO MORTALITY

It was not always easy to attribute the cause of death clearly in these cases. Broncho-

TABLE 4: MORTALITY IN BURNT CHILDREN 0—10 YEARS (1953—1955)
AFRICAN CHILDREN (504 Patients)

Age	Total Deaths	% Mortality	Under 10% Burns			10—15% Burns			15—20% Burns			20—30% Burns			30% + Burns			All Burns over 10%		
			No. of Patients	No. of Deaths	% Mortality	No. of Patients	No. of Deaths	% Mortality	No. of Patients	No. of Deaths	% Mortality	No. of Patients	No. of Deaths	% Mortality	No. of Patients	No. of Deaths	% Mortality	No. of Patients	No. of Deaths	% Mortality
0 to 1 (115)	20	17	97	8	8	10	5	50	5	5	100	2	1	50	1	1	100	18	12	66
1+ to 5 (275)	22	8	230	4	2	27	5	19	6	3	50	8	6	75	4	4	100	44	18	40
5+ to 10 (114)	6	5	97	0	0	9	3	33	3	0	0	2	0	0	3	3	100	17	6	35
Indian Children (74 Patients)																				
0 to 1 (9)	9	33	6	0	0	0	0	0	0	0	0	3	3	100	0	0	0	3	3	100
1+ to 5 (35)	7	20	25	2	8	4	0	0	5	4	80	0	0	0	1	1	100	6	5	83
5+ to 10 (30)	7	23	13	0	0	5	0	0	4	3	75	5	1	20	3	3	100	17	7	41

pneumonia, toxaemia, sepsis and shock were all recorded as the clinical and necropsy diagnoses.

These mortality figures are presented according to age group, and in relation to surface area burnt. No other factors have been taken into consideration, such as the state of nutrition or the time of admission after the accident.

Unquestionably the heavy mortality should be viewed in the light of the general condition of the children before they were burnt. Malnutrition and protein deficiency must play a part, although this has been seldom clinically obvious in the last 40 children admitted, where special note of this was taken. Nevertheless, the general health and nutrition of most of the slum children is poor, and the part played by chronic malnutrition in modifying the body's response to trauma is as yet incompletely assessed.

A major contributory factor to the high mortality was the delay in attending to surgical shock. Whilst the overcrowded conditions and volume of work in such a busy hospital make immediate detailed care difficult, the gravity of the fluid loss and the need for prompt colloid and electrolyte replacement which must be based on an accurate assessment of the burnt area was not always appreciated. Frequently the burn was dressed and an intravenous infusion started some hours later when the patient was in the irreversible phase of shock. The 'extensive' burn was usually well treated initially, as the severity of the injury was appreciated and plasma infusions were supplemented by saline.

It is in the group with 10-30% area burnt that the true assessment of the gravity of the extent was frequently not made, and often termed with little therapeutic significance '2nd degree burns of limb', the critical early hours being allowed to slip away when vigorous treatment promptly applied would have substantially reduced the mortality.

The rate of infection of these injuries was high. Burns must be regarded as large open wounds and only rigorous antiseptic and aseptic care from the moment of admission can reduce the contamination of the potentially infected surface. The application of the dressing, if not properly conducted, too frequently only assists bacterial growth. Unless the toilet and dressing are regarded in the same manner as any surgical exposure is under operating theatre conditions, they are of little value. The problem is rendered difficult in general surgi-

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cal wards where open wounds of all types lie in close proximity with primarily clean burn cases. No burn wound will remain uninfected in such circumstances. Whilst infection played a part in many of the early deaths, it reaped its toll no less frequently in the later days. The ill-nourished child with reserves already depleted had in addition to cope with all varieties of bacterial infection—staphylococci, streptococci, *B. proteus, B. pyocyaneus* and coliform organisms. The profuse exudate leads to a high loss of protein, which further debilitates the patient and helps account for toxaemia, septicaemia and bronchopneumonia, which were noted as causes of death.

Early grafting of such infected areas is rendered useless and the pain of frequent dressings soaked in pus further demoralizes the patient. Whilst awaiting the cleansing of the area by antibiotics, sodden granulations form, early fibrosis advances and by the time grafting can proceed the successes are few and the scarring dense. The hospital stay is prolonged and the area heals slowly, painfully and with contractures, and a multiplicity of failed grafting operations is unavoidable. Finally, the child is left mentally and physically exhausted with the prospect of a long convalescence, rewarded by corrective plastic operations later. This sequence of events is a constant reminder of the near helplessness of antibiotics when first principles of surgical care are hindered by continuous cross-infection.

There are other factors which prevented the reduction of these mortality figures from a level similar to those of Lutken in Copenhagen, 1916-35, quoted by Bull and Fisher,[3] and although they do not feature as visible causes of death, certain of these therapeutic measures have been shown to be detrimental. The use of sulphonamide powder locally on large burn areas presents a danger owing to the rapid absorption rate and the consequent toxicity. Sulphonamide of low percentage in creams specially prepared is usually perfectly safe.

Cortisone was frequently used as an initial treatment, usually in seriously ill cases, often combined with Eucortone, penicillin, streptomycin and sulphonamide powder. Cortisone is dangerous in the early stages of treatment and is contra-indicated in all but a few severe burn cases where there is definite evidence of adrenal failure after some days, and then only after careful selection.[1, 13] Cortisone radically alters electrolyte balance already disturbed and is not of help to a hyperactive adrenal cortex.

The value of daily haemoglobin checks and the judicious use of blood transfusions from the first day onwards is fundamental to treatment. Severe burns succumb rapidly to severe anaemia and in this analysis this must have played a part in both the mortality rate and, equally important, in the failure rate of grafting. Blood was used sparingly and haemoglobin checks were done when clinical evidence of low haemoglobin was apparent. This is unreliable for moderate or even severe drops in haemoglobin levels, and routine estimations should be done at least once or twice weekly or more often in the early stages of treatment. Before undertaking skin grafting it is essential to have an adequate blood volume and a haemoglobin level of not less than 85% in an adult and 75% in a young child.

Finally, nursing has its part to play. Accuracy in urine collection and charting of fluids must be reliable, otherwise treatment becomes largely a matter of guesswork. The nurse must appreciate the need for sympathetic and constant attention, the firmness needed in persuading patients to take their hourly drinks by mouth, the attention to parched, burnt lips and eyelids and the need for the comfort of limbs, buttocks and neck. Above all, there is a real need for mothering these children—the terror of these injuries is as much the fear of the next dressing as the discomfort itself. Any badly treated child will cry uncontrollably as soon as a dressing is touched. The training of nurses for this work should be of no less an order than that for other specialized surgical procedures.

LENGTH OF STAY IN HOSPITAL (TABLE 5)

The average stay of all African children was 18 days, and of Indian children, 26 days, the difference probably being represented by the greater proportion of under 10% burns in the former group. The major burns necessitated a stay of 37 days in African children and 49 days in Indian children, for those who survived the injury. Those children of both races who died averaged 5-7 days in hospital.

The average stay of all surviving children of both races was 21 days. The reason for the low average hospital stay was, in fact, the high mortality rate. Many children were discharged owing to pressure on bed space, to attend as out-patients where granulating wounds were dressed, some to be admitted later for grafting. Major burns over 30% which require many operations and prolonged hospital care did not feature in this series.

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TABLE 5: LENGTH OF STAY IN HOSPITAL

	0 to 1		1+ to 5		5+ to 10		0 to 10
	Number of Patients	*Average Stay in Days*	*Number of Patients*	*Average Stay in Days*	*Number of Patients*	*Average Stay in Days*	*Average Stay of All Age Groups*
African Children (430) Age in Years							
Under 10% area burnt ..	57	22	199	17	78	15	17
Over 10% area burnt and alive	9	31	36	34	16	48	37
Over 10% area burnt and died	12	6	18	3	5	8	5
Total ..	78		253		99		*Overall Average Hospital Stay:— 19 Days*
Indian Children (70)							
Under 10% area burnt ..	5	12	23	15	11	44	23
Over 10% area burnt and alive	1	23	4	51	12	51	49
Over 10% area burnt and died	2	1	6	7	6	9	7
Total ..	8		33		29		*Overall Average Hospital Stay:— 26·5 Days*

because they did not survive; in Becker and Artz' analysis[1] the 20-30% burns stayed in hospital 85.8 days, the 30-40% burns for 124 days and the 40-50% burns for 142 days. Jackson[9] has reported in his series of 1,578 cases of burns that the average healing time for partial thickness burns, i.e. the time from injury to complete healing with all crusts separated, was 20 days; while for full thickness burns the healing time was 51 days. Extensive burns had an average hospital stay of 42-63 days.[9]

THE NEED FOR ORGANIZED CARE OF BURNS

The children in this series were all treated in open surgical wards where cross-infection is inevitable. Colebrook[6] has shown conclusively the value of a burns unit where all possible aseptic precautions are taken, and it is fair to estimate that the establishment of such a unit in hospitals dealing with large numbers of burns would materially reduce the infection, the number of operations and the length of stay.

The cost of these injuries to a hospital is difficult to compute. The cost of treating burns which require multiple operations and large quantities of expensive dressings must be considerably higher than the cost of treating the average surgical patient, and probably at least twice as high. Any improvement in the rate of survival in these children would have meant a considerable increase in the over-all hospital stay. Despite the weighting of the present figures by the preponderance of under 10% burns, if there were an increase of survival to 50% for 40% surface area burns (there is a 50% chance of recovery for 50% burns in almost all major burn centres in Britain and the U.S.A.[1, 11]) which is not unlikely with organized care of burns and skilled nursing, then in a series such as this another 48 African children and 76 Indian children would have survived.

On the basis of Becker and Artz[1] and Jackson's figures,[9] this would mean an additional 2,700 African and 1,234 Indian hospital days. With an increased survival rate and thus an increased length of hospital stay it becomes imperative to try to reduce the length of hospitalization as much as possible. This can best be achieved by such an organized 'unit' where every measure of surgical asepsis can be practised.

The average general hospital that admits 30-50 burns a year should have specially equipped ward side-rooms and dressing rooms for burns treatment, but in hospitals admitting

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more than 100 burns yearly it has become increasingly obvious that there should be a special burns unit. In this hospital where nearly 350 burns cases are admitted yearly, such a unit would be the only method of reducing the high mortality and at the same time prove of economic value to the hospital. Furthermore, many patients with under 10% burns could be treated as out-patients provided trained medical and nursing personnel are available.

In addition to providing an effective centre for therapy, the unit would encourage the establishment of a nucleus of doctors and nurses fully trained in the many specialized techniques of burn shock treatment, control of infection and nutritional and nursing care.

A burns unit is no more than a central ward unit isolated from the established sepsis and, continuous cross-infection of general surgical wards, together with a peripheral part in the Admitting Department where every burn case is taken immediately on arrival. Those not requiring admission are assessed and treated in an aseptic environment. No attendant, doctor, nurse or porter approaches the case without being masked, and any person involved in the dressing should be gowned and use the same no-touch technique as in an operating theatre. With trained staff in this casualty section there would be, after assessment of the major burns, rapid transit to isolation in a cubicle or burns ward where prompt anti-shock treatment would be carried out. A burn by its very nature is sterile at its inception and can be kept so for many hours by the avoidance of frequent inspections alone.[10]

In the central unit each case should preferably be nursed in a separate cubicle, and dressings changed in a fully equipped dressing room or theatre attached to the unit where cross-infection would be kept to a mimimum by planned ventilation and special arrangements for soiled dressing disposal.

The problems of air exchange and control of humidity are refinements—necessary ones —but based on the underlying concept that burns must be treated with rigidly enforced routines of isolation and asepsis. This organization is no more than the routine adopted by units dealing with infectious fevers; it is suggested that it is even more essential for the care of burns which are open wounds, demanding the same care in their inspection and treatment as any recent surgical wound in the ward.

MEANS OF DECREASING THESE ACCIDENTS

A significant reduction in the number of these accidents can be achieved by the family practitioner and child welfare organizations constantly pointing out the dangers to which children are exposed. A child viewing the adult scene from a level of only 2-3 feet cannot be expected to resist pulling down projecting pot or pan handles from the stove, or tugging off an overhanging tablecloth on which jugs of boiling water and tea rest near the edge. Any intelligent mother or nurse-maid will take the precaution of turning handles away from the edge, or avoiding long tablecloths, if she is reminded of the danger. Open fire-grates and unguarded electric elements are potentially lethal and manufacturers, might with advantage be persuaded to comply with minimal safety regulations in supplying fire-guards, as they are in many overseas countries.[7] Colebrook[5-7] has repeatedly pointed out the dangers of highly inflammable cotton or silk clothing which to-day can be substituted by fire-resistant materials. These are important public health aspects which, if dealt with vigorously, would save life and a not inconsiderable drain on hospital resources.

The child's bath can be no less dangerous. Water judged only warm by an adult elbow may cause full thickness loss to a child's delicate skin and addition of boiling water to a lukewarm bath, or cooling water in a tin bath which retains the heat at the bottom, may produce the same result.

A small child left toddling in a main road is exposed to no more danger than if left alone in a kitchen, near a recently vacated breakfast table, or an electric or open fire. The child in the slums is, in addition, at the mercy of overcrowded conditions in small rooms poorly lit by open lamps, and only improved living conditions will materially reduce the accident rate. Despite all the mother's difficulties in such circumstances, timely advice and warning will, nevertheless, make an impression, particularly if she can be made aware of the long months of hospital care and ugly scarring which frequently result from these accidents.

SUMMARY

1. Five hundred and seventy eight cases of burns in children under 10 years admitted to a general hosptial in 3 years have been analysed.

2. The major causes of these injuries are indicated.

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In most cases carelessness is a prominent factor.

There is high proportion of scalds (73%).

3. The age group 1+ to 5 years sustains the greatest number of these injuries.

4. The mortality rates are compared with those which obtain in a large burns unit.

5. Some of the causes for these deaths are discussed.

6. The need for the establishment of a burns unit for centralized and organized care of burns in hospitals dealing with more than 100 burn admissions a year is stressed. It is necessary to separate the cases from the established sepsis of a general surgical ward.

7. The need for strict asepsis is essential as burns should be regarded as open wounds.

8. Some factors which would help reduce these accidents are discussed.

OPSOMMING

1. Vyfhonderd-agt-en-sewentig gevalle van brandwonde by kinders onder 10 jaar wat tot 'n algemene hospitaal oor 'n tydperk van 3 jaar toegelaat is, is ontleed.

2. Die belangrikste oorsake van hierdie beserings word aangedui.

In die meeste gevalle was agtelosigheid 'n belangrike faktor.

'n Groot persentasie van die brandwonde is deur vogtige hitte veroorsaak (73%).

3. Wat hierdie beserings betref, het die grootste aantal by die ouderdomsgroep 1+ tot 5 jaar voorgekom.

4. Die sterftesyfer word vergelyk met dié in eenhede vir die behandeling van groot brandwonde.

5. Party van die oorsake van hierdie sterfgevalle word bespreek.

6. Die noodsaaklikheid vir die daarstelling van 'n brandwonde-eenheid vir die gesentraliseerde en georganiseerde behandeling van brandwonde in hospitale waar meer as 100 slagoffers van brandwonde per jaar behandel word, word beklemtoon. Dit is nodig om hierdie gevalle te skei van die gevestigde sepse van 'n algemene chirurgiese saal.

7. Strenge asepse is noodsaaklik, aangesien brandwonde as oop wonde beskou moet word.

9. 'n Paar van die faktore wat kan help om hierdie soort ongeluk te verminder, word bespreek.

We should like to thank Dr. S. Disler, Medical Superintendent of King Edward VIII Hospital, Durban, for access to the case records and the co-operation of his staff in the task of collecting them; we also thank Mr. C. J. Lockett for diagrammatic material, and Mrs. J. Terry-Lloyd for secretarial work.

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NOTES AND NEWS · BERIGTE

Dr. R. Elsdon-Dew, Director of the Amoebiasis Research Unit, Durban, has given up private practice so as to be able to devote all his time to the expansion of the Unit made possible by the recent grant from the United States Public Health Service.

Dr. G. A. Drummond will carry on the private practice in clinical pathology in Durban.

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Dr. S. Etzine of Johannesburg has been elected a Member of the Foreign Committee of 'Revista Italiana del Tracoma e di Patologia Oculare Virale ed Esotica'.

Dr. M. Cecil Michelow, M.B., B.Ch., D.Obst. R.C.O.G., M.R.C.O.G., who has spent 4 years in the United Kingdom completing his post-graduate training, and who until recently held the post of Assistant Obstetrician and Gynaecologist, Baragwanath Hospital, has joined Drs. S. Joel Cohen and A. M. Porter in specialist obstetric and gynaecological practice at 208 Medical Centre, Jeppe Street, Johannesburg, and 1 Admiral's Court, Rosebank, Johannesburg. (Telephones: *Rooms*: 23-7124, 42-1218; *Residence*: 41-4105).

• • •

Dr. M. Cecil Michelow, M.B., B.Ch., D.Obst. R.C.O.G., M.R.C.O.G., wie vir 4 jaar in Groot Brittanje gewerk het vir verdere opleiding in Ginekologie en Verloskunde, en tot onlangs Assistent Ginekoloog en Verloskundige by Baragwanath Hospitaal, praktiseer tans met drs. S. Joel Cohen en A. M. Porter as spesialiteit in ginekologie en verloskunde by Medical Centre 208, Jeppestraat, en Ad-

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miral's Court 1, Rosebank, Johannesburg. (Telefoon: *Kamers*: 23-7124, 42-1218; *Woning*: 41-4105.

• • •

Prof. S. F. Oosthuizen, President of the South African Medical and Dental Council and at present overseas as a WHO Travelling Fellow, recently accepted an invitation from Sir David Campbell to a dinner of the General Medical Council at 44, Hallam Street, London. Professor Oosthuizen addressed the gathering, which included many prominent persons.

Sir David Campbell, President of the General Medical Council, recently visited South Africa and addressed the South African Medical and Dental Council at the end of its March meeting.

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CRIPPLE CARE: KREUPELSORG

The National Council for the Care of Cripples in South Africa has issued the first number of a new quarterly publication entitled *Cripple Care*: *Kreupelsorg*. It is the official organ of the National Council.

The Editorial Committee consists of Prof. O. Wagner, *Chairman*, Mr. G. T. du Toit, Mr. J. C. Merkin and Dr. I. J. J. van Rooyen, *Secretary*.

Interested medical practitioners can communicate with the National Council for the Care of Cripples in South Africa at P.O. Box 10173, Johannesburg.

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GIBBERELLIC ACID: A NEW PLANT GROWTH STIMULATOR

Gibberellic acid is obtained from cultures of a fungus called *Gibberella fujikuroi*. Chemically it is tetracyclic dihydroxy-lactonic acid, with a formula of $C_{19}H_{22}O_6$.

In past experiments in the Lilly Research Laboratories and elsewhere, gibberellic acid has been shown to stimulate growth in field crops such as wheat, oats, clover, the grasses (including corn) and tobacco. It has also speeded growth in vegetables such as peas, beans, tomatoes, cucumbers and others. It promotes growth in ornamental flowers, shrubs and even trees.

Besides speeding growth, gibberellic acid has other potential uses. In scientific experiments it has been shown to break dormancy in specific plants, to stimulate germination in certain seeds, to reverse dwarfism and to induce flowering of biennials in the year in which they would not ordinarily flower.

Gibberellic acid is much more vigorous in its growth stimulation than another group of plant stimulants known as auxins. It is also more easily controlled. While the auxins inhibit growth at relatively high dosages, comparable dosages of gibberellic acid cause good growth stimulation for extended periods of time.

Extremely minute amounts of the chemical are needed to give plants a growth spurt. In laboratory tests as little as a 0.0000001 g. of gibberellic acid applied to pea seedlings resulted in growth stimulation. The acid was put into solution and applied to the plants with micro-pipettes.

Under controlled conditions, gibberellic acid has been applied successfully in 4 ways: sprayed onto the leaves, fed through the roots, injected into the stem and rubbed onto the stem as part of a lanolin paste.

The effect of this unusual metabolic agent on the animal cell is not yet known.

PENICILLIN DESENSITIZATION

The following discussion appeared under *Queries and Minor Notes* in J. Amer. Med. Assoc., on 9 March 1957:

To the Editor: Is it possible to desensitize people to penicillin? R. J. Sexton, M.D., Charleston, W.Va.

Replies from two consultants were received as follows:

Answer: There is no practical reliable method of desensitizing penicillin-sensitive patients. The fractional method of desensitization, even where effective, would take too long to be of any practical value. This procedure is not without danger in patients anaphylactically or atopically sensitive to penicillin. It is suggested that some other suitable antibiotic be substituted where penicillin sensitivity is suspected.

Answer: Desensitization to penicillin has been reported following the use of various techniques. However, it is difficult to evaluate these procedures since it is not uncommon for a sensitive patient to become spontaneously desensitized. A patient in whom further treatment is indicated, as in syphilis, may develop urticaria after the institution of penicillin therapy, but often continued administration of this antibiotic fails to cause an exacerbation of the hives. Furthermore, as treatment is continued the reaction is apt to subside. Especially with the use of antihistaminics and corticosteroids, practically any such patient can be treated with penicillin to a successful completion of his course of therapy.

• • •

PALLIATION OF OVARIAN CARCINOMA WITH PHOSPHORAMIDE DRUGS

Observations on the treatment of far advanced ovarian carcinoma with phosphoramide drugs are reported.

Twenty-two patients were treated for periods of 1-22 months with total doses of 110-780 mg. (average 353 mg.) of N-N′-N″ triethylene thiophosphoramide. Short periods of substitution therapy with N-3 (oxapentamethylene) N-N′-diethylene phosphoramide were used in 8 cases.

Routes of therapy included intravenous, intrapleural, intraperitoneal, intrapericardial, intratumour, transvaginal and transabdominal. In 2 cases abdominal masses were injected during laparotomy.

Objective improvement included regression of tumour masses and control of pleural, pericardial and peritoneal effusions.

Ten patients were alive 1-22 months following institution of therapy; of 12 who died or were lost to follow-up, 8 showed improvement for periods ranging from 1-11 months.

Therapy with phosphoramide drugs appears to be palliative only and must be continued to achieve and maintain control of disease.

The observations reported by these workers* appear to warrant further therapeutic trials with triethylene thiophosphoramide in ovarian carcinoma.

* Bateman, J. C. and Winship, T. (1956): Surg., Gynec. Obstet., **102**, 347.

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British Bursary for Post-Graduate Clinical Study, P.O. Box 1010, Johannesburg.

The closing date for applications is 31 July 1957.

SELECTION COMMITTEE

The following have agreed to serve on the Selection Committee: Prof. G. A. Elliott, Prof. F. Forman, Prof. S. F. Oosthuizen, Dr. H. A. Shapiro, Dr. M. Shapiro, Dr. M. M. Suzman.

REVIEWS OF BOOKS

THE TREPONEMATOSES

Biology of the Treponematoses. By Thomas Bourne Turner and David H. Hollander. Geneva, 1957 (*World Health Organization: Monograph Series,* No. 35). (278 pages. Price £1 10s.)

In 1950 the International Treponematosis Laboratory Center was established in the Department of Microbiology of the Johns Hopkins University under the joint auspices of WHO and the Johns Hopkins School of Hygiene and Public Health.

From its inception WHO has encouraged and assisted the work of the Center. Largely as the result of these investigations a picture of the treponematoses now emerges which differs from that of earlier years inasmuch as the treponemal diseases have now been brought more closely in line with other infectious processes. It is the observations and principles underlying these changing concepts that provide some of the most important material included in the *Biology of the Treponematoses.*

The monograph is arranged in three parts. Part I deals with the biology of treponemal infections and includes chapters on the sources of strains used during the investigations, the experimental disease in laboratory animals, factors affecting its evolution, characteristics of treponemes *in vitro,* immunity phenomena, and the response of treponemes to drugs. Part II, which comprises a comparative study of treponemes, discusses the characteristics of the experimental disease invoked by various strains isolated from different parts of the world, the immunological relationships between these strains; and their behaviour in response to penicillin. Part III consists of a single chapter providing a useful summary of the principal findings set forth in preceding sections.

Two appendices are included. The first gives details of the isolation of strains, each strain being designated according to its geographical source, while the second supplies information on the prevention and treatment of laboratory accidents that may occur during the course of experimentation.

Although much has been accomplished by these studies, several problems await elucidation. The authors of the monograph suggest a number of fields that lie open to the investigator, in the hope that the knowledge to be gained from such research will aid in the ultimate conquest of the treponematoses.

CEREBRAL PALSY

Cerebral Palsy. By Cyril B. Courville, M.D. (Pp. 77 + Index. With 28 Figs. 1954). Los Angeles: San Lucas Press.

In a book of 80 pages the author has given an outline of the incidence, aetiology, physiology, pathology, clinical syndromes, diagnosis, treatment and prognosis of cerebral palsy.

As cerebral palsy is a very specialized field, a book written for the medical specialist or the therapist working in this field would be greatly appreciated. The contents of this book do not fulfil this function.

The author is generous in accepting the beneficial effects alleged for numerous drugs in this condition, and also the part that operative intervention on the part of neurosurgeons and orthopaedic surgeons can play in cerebral palsy. According to most experts, surgery and drugs are of very limited value. The author's own experience with these drugs and operations is not mentioned.

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The book is disappointing as there is no practical guidance for the occupational therapist, the speech therapist, the physiotherapist or the teacher working in the cerebral palsy field.

PSYCHO-SEXUAL PATHOLOGY

The Psychology of Sex Offenders. By Albert Ellis, Ph.D., and Ralph Brancale, M.D. With the collaboration of Ruth R. Doorbar. 1956. (Pp. 132. 27s. 6d.). Oxford: Blackwell Scientific Publications Limited.

Sexual pathology is a field dominated more by opinion than observations and, in our society, a moralistic statement frequently does duty for a fact. In such a climate it is difficult for any would-be investigator to avoid being either a puritan purveying *ex cathedra* statements or a free thinker who does not believe a sex offender exists. The authors of this tidy statistical study have avoided both temptations, and their book is a neat collection of simple facts about 300 convicted male offenders. It is heartening, if not surprising, to find the convicted offender is rarely psychotic; that, though disturbed, he is no more likely to be a gross neurotic than any other offender and that, far from being sexually active, he tends to be more inhibited and sexually incompetent than his fellows. As for the fashionable belief that childhood rejection is responsible for all ills, only 45% showed evidence of such deprivation and of the remainder there was a large number showing evidence of over-protection in childhood. The material shows that the present classification of sex offenders used in the courts in New Jersey has little relationship to the scientific classification—a finding which would probably be true in this country.

Though the statistical study is illuminating, and the proposals the authors put forward for the revision of the law concerning sex offenders are well considered, the other sections of the book are scrappy and could have well been either omitted altogether or else expanded. The chapter on interrogation of sex offenders adds little to the theory of interviewing and says little that any well-trained interviewer does not already know. The discussion of normal sex behaviour is very brief and, though one must agree with it all, others have dealt with the question more fully; but perhaps these chapters (and maybe the whole of the book) are intended to be read by those dealing with sex offenders but who themselves have had no psychological training. If this is the case, then their inclusion is justified. The book, in any case, is one that social workers, lawyers and probation officers would be well advised to read; it would encourage a more liberal attitude than is evident in the customary approach to sexual crime and would help to remove some of the superstitions which riddle the minds of most of us.

The book is well produced, and is a model of those high standards of book design which are coming to be a matter of course in publishing.

CORRESPONDENCE

PREVENTION OF BLINDNESS IN THE BANTU

To the Editor: The report on the prevention of blindness published in your issue of 25 May reminds me of my experiences with trachoma in the Cape Province.

When I was Acting Matron at the Athlone School for the Blind (1931-1937) the late Dr. Wood entrusted me with the application of copper sulphate to 6 and later to many other cases diagnosed by him as trachoma. These patients attended the Out-Patients' Department at the New Somerset Hospital, Green Point.

I clearly remember his diagnosis of trachoma because some of his colleagues were not in agreement with him. These cases were all Cape Coloureds coming from the Cape Flats near Maitland and Zoar, near Ladismith, Cape.

Florence M. Blaxall.

P.O. Box 33,
Heidelberg, Tvl.

PREVENTION OF PENICILLIN REACTIONS

To the Editor: On reading Dr. Davidson's excellent article on the prevention of penicillin reactions in your issue of 2 March 1957, I noticed that he refers to one recorded case of an immediate anaphylactic reaction following the use of penicillin ointment in the eye.

It would seem that penicillin reactions are becoming more common and dangerous, and I feel it is worth while stressing the danger of using penicillin indiscriminately even as eye drops, as I have seen a case of anaphylaxis following instillation of penicillin eye drops into the conjunctival sac.

The patient, who had a foreign body in the cornea of his only eye (the left eye), reported during August 1956; 1-2 drops of Pontocaine 1% were instilled and a large embedded corneal foreign body was removed under the corneal microscope. This was followed by the instillation of a few penicillin drops. About 15-20 minutes later the patient (who fortunately was still in the building) reported, saying that he felt queer and within minutes was in a state of acute anaphylactic shock. He recovered after intensive treatment, and I am convinced he would have died had he not been given immediate treatment.

It is possible that the anaphylaxis followed the instillation of Pontocaine.

He has since had another foreign body removed under xylocaine local anaesthesia without incident, but, needless to say, this was not followed by the instillation of penicillin into the eye.

R. A. Trope.

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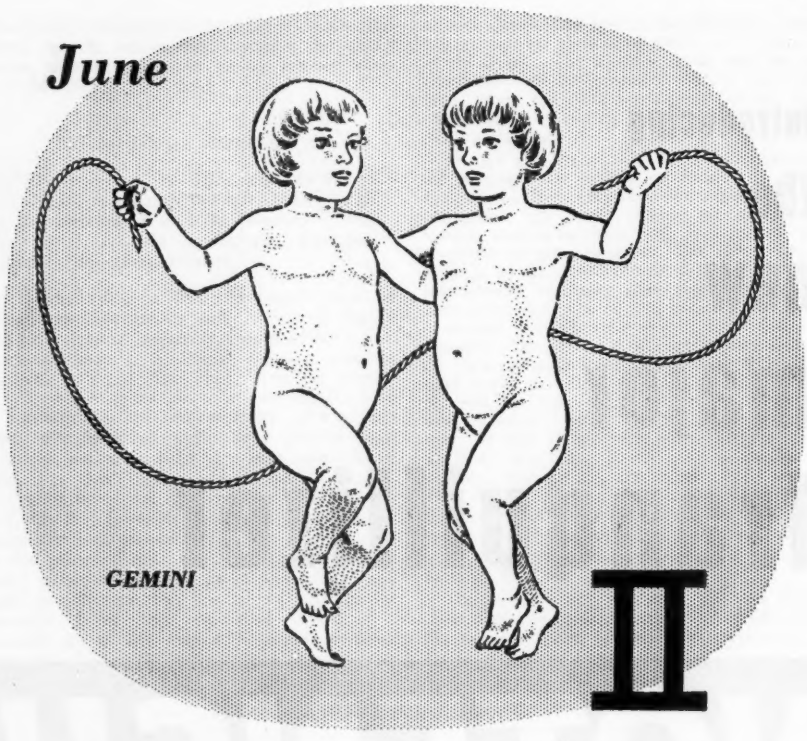

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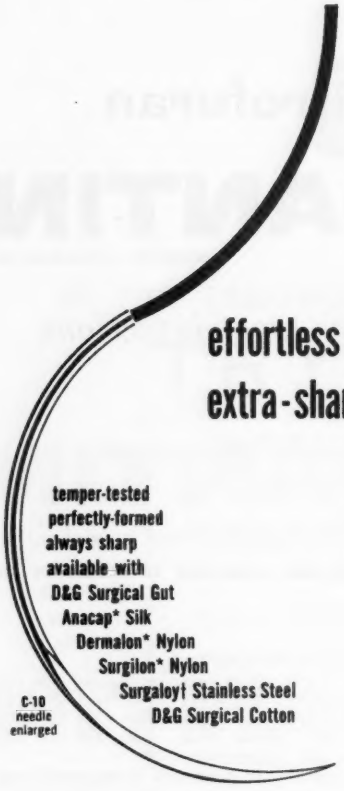

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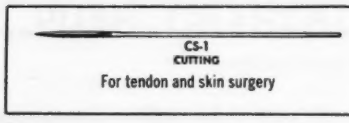

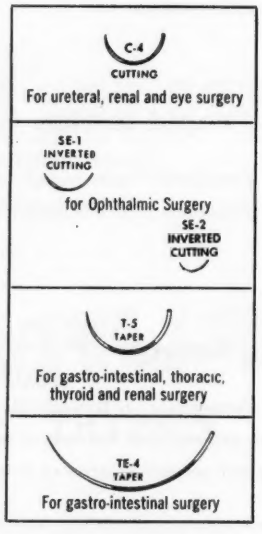

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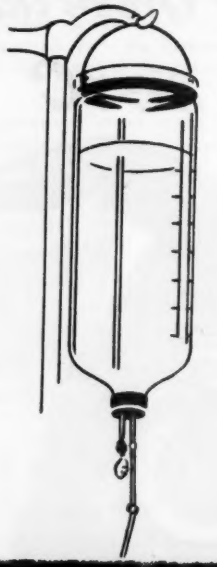

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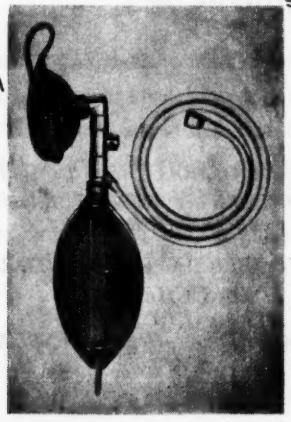

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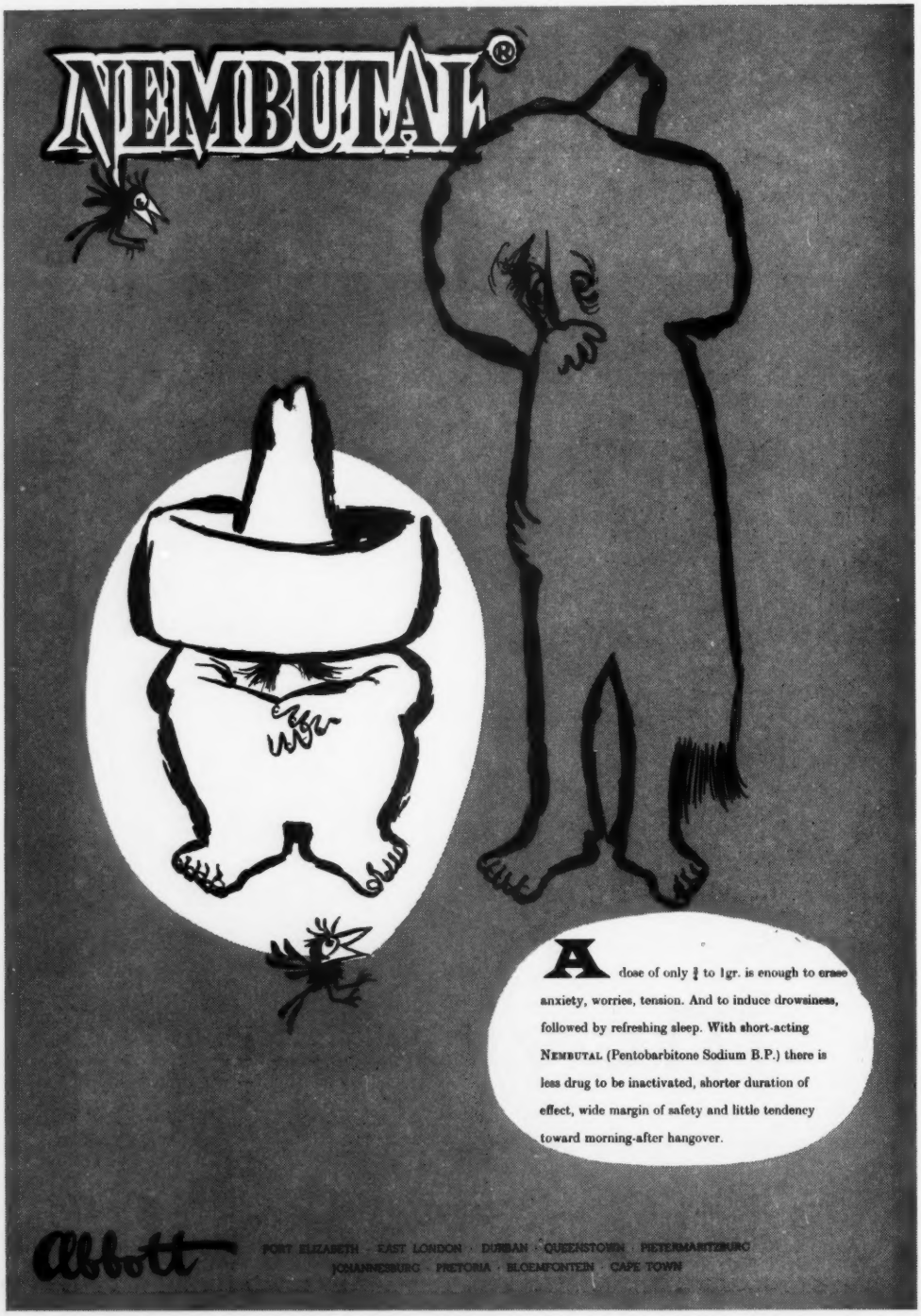
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Published by the Proprietors Juta and Co. Ltd., 43 Pritchard Street, Johannesburg, and printed in the Union of South Africa by Cape Times Limited, Parow, C.P.